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ARCHIVES OF SURGERY

VOLUME 24 JANUARY, 1932 NUMBER 1

THE ROENTGENOLOGIC DISPLACEMENTS IN COLLES' FRACTURE

WITH SPECIAL REFERENCE TO THE MECHANISM OF THE ACCOMPANYING FRACTURE OF THE ULNAR STYLOID: A REPORT OF ONE HUNDRED CONSECUTIVE CASES *

LOUIS CARP, M.D.

NEW YORK

This study was undertaken to show: (1) the usual roentgenologic displacements in Colles' fracture with an accompanying fracture of the styloid process of the ulna and the practical therapeutic value of such information, and (2) the probable mechanism of fracture of the ulnar styloid. According to Schinz,[1] this fracture occurs in 58 per cent of all Colles' fractures.

A study of the table will demonstrate the most frequent positions of the lower radial fragment (Darrach [2]) in this series of cases, as follows:

	Percentage
Impaction only	29
Dorsal tilt with impaction	17
Dorsal tilt with dorsal displacement	12
Dorsal tilt with dorsal displacement and impaction	12
Dorsal tilt	9
Simple fracture, no displacement	4
Dorsal tilt, dorsal displacement, radial shift	4
Ventral tilt with impaction	3
Dorsal displacement with impaction	3
Dorsal tilt, impaction, radial shift	2
Dorsal displacement	2
Ventral tilt, ventral displacement, impaction	1
Dorsal tilt, dorsal displacement, radial shift	1
Dorsal tilt, radial shift	1

From these statistics it may be inferred that, generally speaking, Colles' fractures with accompanying ulnar styloid fractures have enough roentgenologic displacements to warrant their reduction. Experience has taught that lack of gross deformity does not necessarily mean a lack

* Submitted for publication, Feb. 24, 1931.

* From the Fracture Clinic of Dr. William Darrach, the Presbyterian Hospital, and the College of Physicians and Surgeons, Columbia University.

1. Schinz, Hans R.: Der Abbruch des Processus styloidens ul..ae, Deutsche Ztschr. f. Chir. **125**:81, 1922.

2. Darrach, William: Fractures of the Lower Extremity of the Radius; Diagnosis and Treatment, J. A. M. A. **89**:1683 (Nov. 12) 1927.

Analysis of Roentgenologic Findings in One Hundred Consecutive Cases of Colles' Fracture with Accompanying Fracture of the Ulnar Styloid

Fracture of Radius	Fracture of Ulnar Styloid																				
	Base				Middle				Tip				Base and Middle				Base and Tip				
	Shift†		Angulation†		Shift		Angulation		Shift		Angulation		Shift		Angulation		Shift		Angulation		
	Lateral	Mesial	Lateral	Mesial	Lateral	Mesial	Lateral	Mesial	Lateral	Mesial	Lateral	Mesial	Lateral	Mesial	Lateral	Mesial	Lateral	Mesial	Lateral	Mesial	Total
Impaction only	—	—	+	—	+0.3	—	—	—	+0.2	—	—	—	..	..	..	..	..	..	..	..	
	—	—	+45	—	—	—	—	—	+0.2	—	—	—	..	..	..	..	..	..	..	..	
	—	—	—	—	+0.1	—	—	—	—	—	—	—	..	..	..	..	..	..	..	..	
	—	—	—	—	+0.3	—	+20	—	—	—	—	—	..	..	..	..	..	..	..	..	
	—	—	—	—	+0.2	—	—	—	—	—	—	—	..	..	..	..	.	..	..	..	
	—	—	—	—	+0.1	—	—	—	+0.1	—	—	—	..	..	..	..	..	..	..	..	
	—	—	—	—	—	—	—	—	—	—	—	—	..	..	..	..	..	..	..	..	
	—	—	—	—	+0.1	—	—	—	+0.5	—	+80	—	..	..	..	..	..	..	..	..	
	—	—	—	—	—	—	—	—	—	—	—	—	..	..	..	..	..	..	..	..	
	—	—	+10	—	..	..	..	..	—	—	—	—	..	..	..	..	..	..	..	..	20
Dorsal tilt, impaction	—	—	+35	—	+0.2	—	+10	—	—	—	—	—	—	—	—	—	..	..	..	..	
	+0.3	—	—	—	—	—	—	+	—	—	—	—	..	..	..	..	..	..	..	..	
	+0.2	—	+20	—	+0.3	—	+10	—	+0.5	—	+45	—	..	..	..	..	..	..	..	..	
	—	—	—	—	+0.1	—	—	—	..	..	..	..	..	..	..	..	..	..	..	..	
	—	+	—	—	+0.3	—	—	—	..	..	..	..	..	..	..	..	..	..	..	..	
	+0.05	—	—	—	—	—	—	—	..	..	..	..	..	..	..	..	..	..	..	..	
	—	—	—	—	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	17
Dorsal tilt, dorsal displacement, impaction	—	—	+10	—	+0.2	—	—	—	+0.3	—	+110	—	..	..	..	..	+0.1	—	+10	—	
	+0.1	—	—	—	+0.3	—	+10	—	—	—	—	—	..	..	..	..	+0.05	—	—	—	
	—	—	—	—	+	—	—	—	..	..	..	..	..	..	..	..	..	..	..	..	
	—	—	—	—	+0.5	—	+90	—	..	..	..	..	..	..	..	..	..	..	..	..	12
Dorsal tilt, dorsal displacement, no impaction	+	—	—	—	+0.5	—	—	—	+	—	—	—	..	..	..	..	..	..	..	..	
	+0.3	—	—	—	+0.3	—	+	—	—	—	—	—	..	..	..	..	..	..	..	..	
	—	—	—	—	—	—	—	—	..	..	..	..	..	..	..	..	..	..	..	..	
	+0.2	—	—	—	—	—	—	—	..	..	..	..	..	..	..	..	..	..	..	..	
	+0.2	—	—	+10	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	
	—	—	—	—	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	12

Dorsal tilt	—	—	—	—	+0.2	—	+	—	+0.2	—	—	—	..	..	..	..	..	..	..	..	
	?	..	..	..	+0.1	—	—	—	..	..	..	..	..	..	..	..	..	..	..	..	
	+0.3	—	—	—	—	—	—	—	..	..	..	..	..	..	..	..	..	..	..	..	
	+0.4	—	—	—	—	—	—	—	..	..	..	..	..	..	..	..	..	..	..	..	9
Simple fracture*	—	—	—	—	+0.3	—	—	—	—	—	—	—	..	..	..	..	..	..	..	..	
	—	—	—	—	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	4
Dorsal tilt, dorsal displacement, impaction, radial shift‡	—	+0.1	—	—	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	
	—	—	—	—	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	
	+0.1	—	+10	—	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	
	+0.1	—	+10	—	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	4
Ventral tilt, impaction	—	—	+110	—	..	..	..	..	+	—	—	—	..	..	..	..	..	..	..	..	
									+0.2	—	+45	—	..	..	..	..	..	..	..	..	3
Dorsal displacement, impaction	—	—	+10	—	—	—	—	—	+	—	—	—	..	..	..	..	..	..	..	..	3
Dorsal displacement	—	+6.1	—	—	..	..	..	..	+0.2	—	—	—	..	..	..	..	..	..	..	..	2
Dorsal tilt, impaction, radial shift	—	—	—	—	..	..	..	..	..	..	..	..	+1.2	—	+20	—	..	..	..	..	2
Ventral tilt, ventral displacement, impaction	..	..	..	..	..	..	..	..	+0.3	—	—	—	..	..	..	..	..	..	..	..	1
Dorsal tilt, dorsal displacement, radial shift	—	—	—	—	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1
Dorsal tilt, radial shift	—	—	—	—	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	..	1
Total.....	43				29				24				2				2				100

* No displacement or impaction.
† The distance of shifts is measured in centimeters. Angulation is measured in degrees; + indicates present; —, absent.
‡ Radial shift is measured from the lesser sigmoid cavity of the radius.

of roentgenologic displacement. Since, in Colles' fracture in particular, roentgenologic fragment reduction of the radius is so important in the majority of cases for production of good anatomic and functional end-results, the foregoing statistics indicate the necessity of careful study of the roentgenogram before reduction or immobilization is attempted.

THE MECHANISM OF FRACTURE OF THE ULNAR STYLOID

It is my belief that in the greatest percentage of cases the ulnar styloid is fractured at the base by a sudden pull of the intra-articular fibrocartilage and at the middle and tip by a sudden pull of the ulnar collateral ligament, and that both ligaments may act together to produce the aforementioned fractures. A small percentage of these fractures may be produced by a sudden impact of the carpus against the ulnar styloid.

In order that these theories might be substantiated, the problem was approached from the following angles:

1. An anatomic study was made of the ligamentous attachments to the ulnar styloid to determine lines of stress and strain with respect to the accompanying fracture of the radius and the other bony structures. It was felt that experimental production in the cadaver of the fractures under discussion would be difficult, and that any definite conclusions would be unwarranted (Cotton,[3] Gallois [4]).

2. A notation was made of the sites of fracture of the styloid process, with their various displacements, as shown by the roentgenograms (figs. 1, 2 and 3).

3. The positions and displacements of the lower radial fragment, as shown anatomically and roentgenologically, were correlated.

ANATOMIC STUDY OF LIGAMENTS ATTACHED TO THE ULNAR STYLOID

The wrist joints of twenty-two cadavers were dissected in order to note especially the ligamentous attachments to the ulnar styloid (fig. 4). The two chief structures that produce stress and pull on the styloid are the intra-articular triangular fibrocartilage and the ulnar collateral (internal lateral) ligament.

The Triangular Fibrocartilage.—This ligamentous attachment is triangular, its base being joined to the mesial portion of the articular surface of the lower end of the radius and its apex to the notch at the

3. Cotton, F. W.: Experimental Colles's Fracture, Boston Soc. M. Sc. **2**:171, 1897-1898.

4. Gallois, E.: Fracture de l'extrémité inférieure du radius; étude radiographique, physiologique et expérimentale, Lyon, L. Bourgeon, 1898.

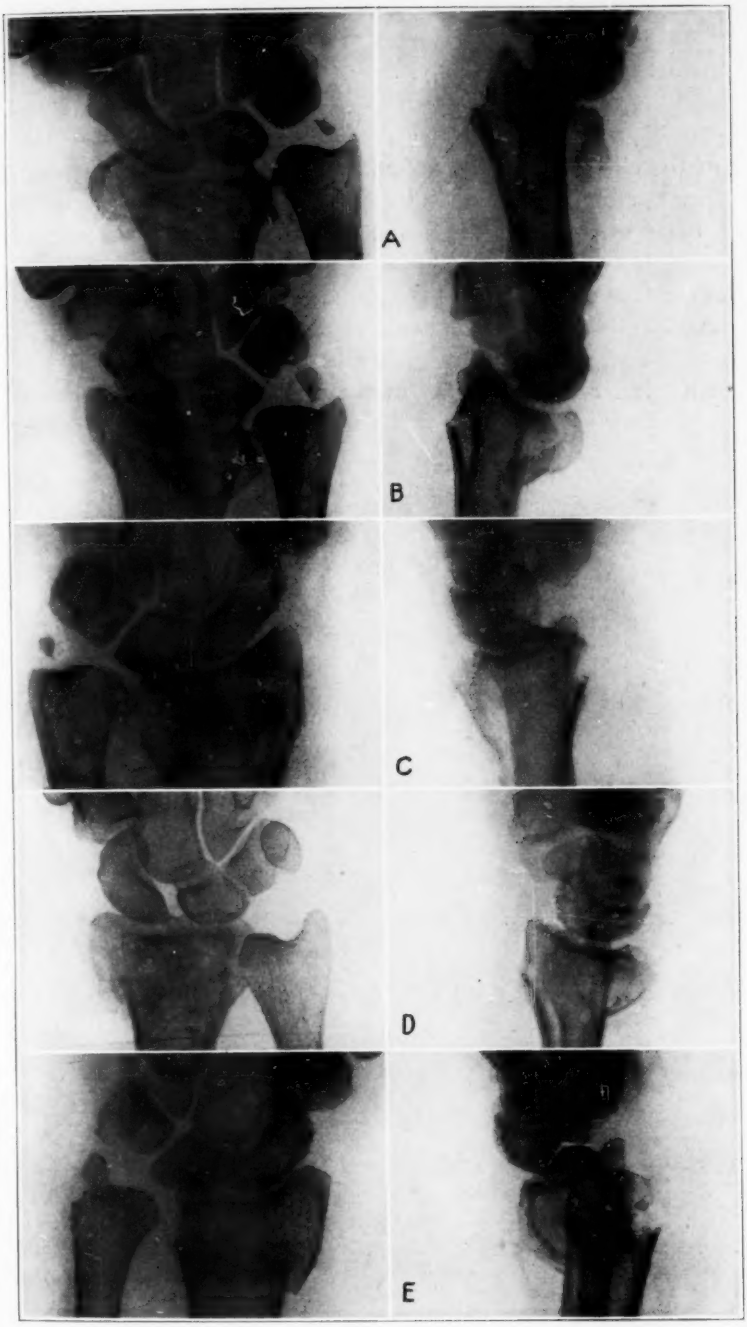

Fig. 1.—Types of fractures of the ulnar styloid accompanying Colles' fracture probably produced by pull of the intra-articular fibrocartilage: *A*, dorsal tilt, dorsal displacement, impaction; fracture of the middle of the ulnar styloid with lateral shift of 0.5 cm. and angulation of 90 degrees. *B*, dorsal tilt, dorsal displacement, impaction; fracture of the base of the ulnar styloid, with lateral shift of 1 cm. and angulation of 10 degrees. *C*, dorsal tilt, dorsal displacement, impaction, dorsal free fragment; fracture of the middle of the ulnar styloid with slight lateral shift. *D*, dorsal tilt, dorsal displacement, radial shift; fracture of the base of the ulnar styloid without displacement. *E*, dorsal tilt, dorsal displacement, impaction, radial shift; fracture of the base of the ulnar styloid with angulation of 45 degrees.

base of the ulnar styloid. It articulates both bones firmly. Its anterior and posterior borders are united with the anterior and posterior radio-ulnar ligaments. Both surfaces are covered by synovial membrane. The following additional observations were noted:

1. The triangular fibrocartilage is about 1.1 cm. long at its base and the same length from apex to base.

2. It is very strong at its periphery and becomes thinner at its center and at the lesser sigmoid cavity of the radius. In three specimens, the thin central portion was found to be completely eroded. It is difficult to say whether this occurred ante mortem or post mortem.

Fig. 2.—Types of fracture of the ulnar styloid accompanying Colles's fracture, probably produced by pull of the ulnar collateral ligament: *A*, ventral tilt with impaction; fracture of the tip of the ulnar styloid with lateral shift of 0.2 cm. and lateral angulation of 45 degrees. *B*, impaction; fracture of the tip of the ulnar styloid without shift or angulation. *C*, dorsal tilt, impaction; fracture of the tip of the ulnar styloid without shift or angulation.

3. The reinforcements of the anterior and posterior radio-ulnar ligaments are important structures.

4. The central fibers form a thick, strong strand of fibrous tissue which attaches itself intimately in an arched direction to the notch at the base of the ulnar styloid. Up to this point the fibrocartilage does not attach itself to the ulna.

5. In sixteen of the twenty-two specimens, additional weaker fibers of the fibrocartilage attached themselves along the entire radial border of the ulnar styloid.

6. In five specimens, additional weaker fibers of the fibrocartilage attached themselves along only a part of the radial border of the ulnar styloid.

7. In one specimen, the fibrocartilage inserted only in the notch at the base of the ulnar styloid.

8. There is a small blood vessel that enters the arched fibers of the fibrocartilage. It seems to come from the bone.

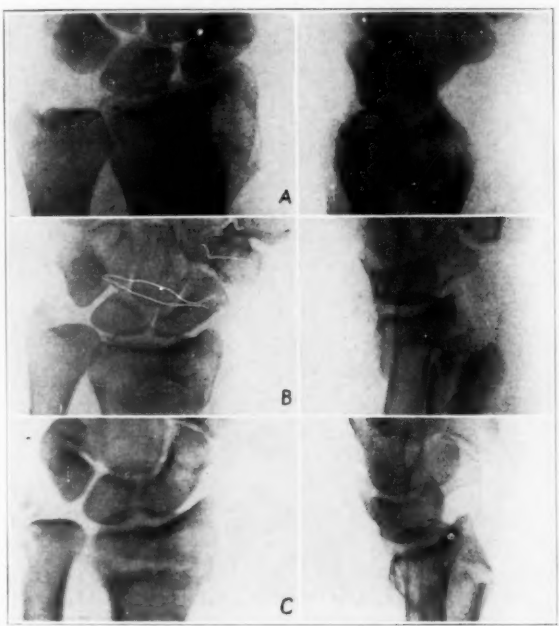

Fig. 3.—Types of fractures of the ulnar styloid accompanying Colles' fracture, probably produced by a combined pull of the intra-articular fibrocartilage and ulnar collateral ligament: *A*, dorsal tilt, impaction and radial shift; fractures of the base and middle of the ulnar styloid with lateral shifts of the fragments of 0.1 and 0.2 cm., respectively, and angulation of 20 degrees. *B*, dorsal tilt, dorsal displacement, impaction; fractures of the base and tip of the ulnar styloid, with lateral shift of 0.5 cm. *C*, dorsal tilt impaction; fractures of the base, middle and tip of the ulnar styloid.

The function of the triangular fibrocartilage is to cushion the carpus against the radio-ulnar joint and to limit pronation and supination at the wrist joint.

The Ulnar Collateral (Internal Lateral) Ligament.—This is a strong, fan-shaped, cordlike structure the apex of which is attached to

the ulnar styloid. From the apex it spreads out and divides into an anterior and posterior fasciculus; the former, passing down vertically, is attached to the inner side of the cuneiform bone, the latter to the base of the pisiform bone and annular ligament. The following additional notations were made from the dissections:

1. The tendon of the extensor carpi ulnaris, by grooving the ulnar collateral ligament on its dorsal surface, throws up the fibers mesially in a thick, cordlike structure which passes mostly to the tip of the styloid process.

2. The anterior fasciculus spreads out along the radial border of the ulnar styloid and from there continues up to the ulnar head.

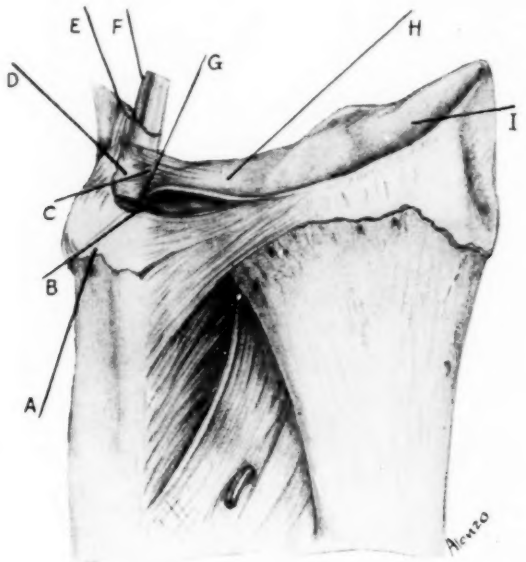

Fig. 4.—Drawing from a dissection to show the anatomy of the intra-articular triangular fibrocartilage and the ulnar collateral ligament. *A* indicates the anterior fibers of the ulnar collateral ligament; *B*, the thickened arcuate fibers of the triangular fibrocartilage inserting into the notch at base of ulnar styloid; *C*, additional fibers of the triangular fibrocartilage inserting into the radial surface of the ulnar styloid; *D*, the styloid process of the ulna; *E*, the posterior fibers of the ulnar collateral ligament thrown into a strong fold by a tendon of the extensor carpi ulnaris; *F*, the tendon of the extensor carpi ulnaris; *G*, the ulnar articular surface; *H*, the triangular fibrocartilage (note the thin central portion) and *I*, the articular surface of the radius.

3. The fibers of the anterior fasciculus nearer the joint surface have a tendency to insert on the radial aspect of the ulnar styloid if the triangular fibrocartilage is not attached all along its length.

The action of the ulnar collateral ligament is to limit pronation, supination, extension and abduction of the wrist.

ROENTGENOLOGIC FINDINGS

The following is a summary of the analysis presented in the table:

1. Almost half of the fractures of the ulnar styloid accompanying Colles' fracture occur at its base.

2. In all the fractures of the ulnar styloid, including the base, middle and tip, 29 per cent occurred with impaction only of the radius fracture.

3. When the alinement between the lower radial fragment and the shaft of the radius was disturbed, there was in the greatest percentage of cases lateral shift, lateral angulation or both in the fractured ulnar styloid.

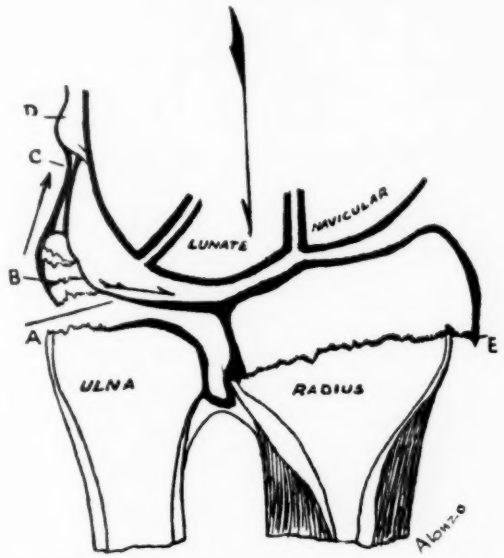

Fig. 5.—Diagrammatic view of a vertical section of the wrist showing the probable mechanism of fracture of the base of the ulnar styloid by the intra-articular fibrocartilage and of the middle and tip of the ulnar styloid by the ulnar collateral ligament (arrows show direction forces). *A* indicates the triangular fibrocartilage; *B*, the ulnar styloid; *C*, the ulnar collateral ligament; *D*, the pisiform bone, and *E*, the line of fracture.

4. With impaction only of the radius accompanied by fracture of the middle of the ulnar styloid, there was lateral shift of the styloid fragment in two thirds of the cases; accompanied by fracture of the tip, there was lateral shift in two fifths of the cases; accompanied by fracture of the base, there was no instance of shift.

5. Lateral shift of the ulnar styloid is more than twice as frequent as lateral angulation.

6. Medial shift and medial angulation of the ulnar styloid are rare.

7. Fracture of the ulnar styloid accompanying simple fracture (no displacement or impaction) of the lower end of the radius is relatively infrequent. There were only four cases in the series.

CORRELATION OF THE ANATOMIC AND ROENTGENOLOGIC FINDINGS

Mechanism of Fracture of the Base of the Ulnar Styloid (fig. 5).—The relation of the intra-articular fibrocartilage to the ulnar styloid and the pull it exerts from the radial fragment make it logical to assume that in the greatest percentage of cases the pull on the ulnar styloid by the fibrocartilage produces a fracture at its base. This pull is produced by a forceful impact of the carpus against the fibrocartilage and by a sudden strain on the fibrocartilage by the displaced lower radial fragment. Part of the ligamentous pull may be spent by a tear in the ligament.

Roentgenologic Findings in Support of Foregoing Theory.—Impaction plus tilts plus displacements of the radial fragment (or their combinations) produced forty-one fractures of the base of the ulnar styloid. In these forty-one fractures there were twenty-one lateral and three medial shifts, ten lateral and one medial angulations.

The great preponderance of lateral shifts and angulations of the styloid process seems to indicate a pull by the fibrocartilage from its point of origin on the radius.

Mechanism of Fracture of the Middle of the Ulnar Styloid (fig. 5).—The mode of attachment of the ulnar collateral ligament seems to make it the logical factor for the production of fracture of the ulnar styloid at this site in the greatest percentage of cases. The dissociation of the anatomic relations at the wrist joint, especially with a medial shift of the carpus, tends to put undue tension on the ulnar collateral ligament. Part of the ligamentous pull may be spent by a tear in the ulnar styloid, or both.

Roentgenologic Findings in Support of Foregoing Theory.—Impactions plus tilts plus displacements of the radial fragment (or their combinations) produced twenty-eight fractures of the middle of the ulnar styloid. In these twenty-eight fractures there were eighteen lateral shifts and seven lateral and one medial angulations.

Mechanism of Fracture of the Tip of the Ulnar Styloid (fig. 5).—It is logical to assume that the ulnar collateral ligament is responsible for the largest percentage of fractures of the tip of the ulnar styloid because of the way it is attached to this bone.

Roentgenologic Findings in Support of Foregoing Theory.—Impactions plus tilts plus displacements of the radial fragment (or their com-

binations) produced twenty-three fractures of the tip of the ulnar styloid. In these twenty-three fractures there were thirteen lateral shifts and four lateral angulations.

Mechanism of Fracture of the Base and Middle or Base and Tip of the Ulnar Styloid.—This type of fracture is probably produced by a combined action of the fibrocartilage and ulnar collateral ligament.

Roentgenologic Findings in Support of Foregoing Theory.—Impactions plus tilts plus displacements of the radial fragment (or their combinations) produced two fractures of the base and middle of the ulnar styloid. In these two fractures there were one lateral shift and one lateral angulation.

Impactions plus tilts plus displacements of the radial fragment (or their combinations) produced two fractures of the base and tip of the ulnar styloid. In these two fractures there were two lateral shifts and one lateral angulation.

Fracture of the ulnar styloid by direct violence is necessarily rare because it is such a small bony process, very well protected by soft structures. It is likely that fractures that have a medial shift or angulation may be broken by direct forceful impact of the carpus against the styloid process.

SUMMARY

1. Most Colles' fractures accompanied by fractures of the ulnar styloid have sufficient (96 per cent) displacements of the lower radial fragment to warrant reduction in order to obtain the best possible anatomic realinement which is usually essential for the best end-results.

2. A correlation of roentgenologic and anatomic findings suggests that the ulnar styloid in Colles' fracture, in the great majority of cases, is fractured at the base by the pull of the intra-articular fibrocartilage of the wrist joint and at the middle and tip by the pull of the ulnar collateral ligament. It is logical to assume that both ligamentous structures may act together to produce fracture of the ulnar styloid, especially when the fracture occurs in two or three places simultaneously. Direct violence probably plays a negligible rôle in any of the fractures of the ulnar styloid, although forceful sudden impact of the carpus against the styloid may be the cause in a very small percentage of the cases.

QUOTATIONS FROM THE LITERATURE ON MECHANISM OF FRACTURE OF THE ULNAR STYLOID IN COLLES' FRACTURE

I believe that there are two factors at work: the internal lateral ligament which tears off the tip and the triangular fibrocartilage which produces fracture near the base. (Fullerton [5])

5. Fullerton, A.: Colles's Fracture, M. Press **79**:555, 1905.

The fragment of the ulnar styloid is usually so small that it is torn away by a twist of the internal lateral ligament at its point of insertion. (Hennequin [6])

With the displacement of the radius either the ligaments must rupture or the ulnar styloid must be broken. (Duplay [7])

If the hand is in adduction the semilunar bone receives the whole weight and the triquetral becomes approximated to the ulna, the styloid process of which may be torn by pressure on the fibrocartilage. In hyperextension the styloid may be torn either by pressure of the semilunar on the triangular fibrocartilage or by traction of the internal lateral ligament. (Gallois [4])

When the radius breaks and the fracture forces continue to act, the hand is forcibly turned inwards and upwards, thus putting the cord-like internal lateral ligament on the stretch and in a test of strength between the ligament and the bone, the latter gives way. (Van Lennep [8])

The displacement of the radius may produce fracture of the ulnar styloid (65%), rupture of the fibrocartilage or internal lateral ligament or combinations of these. If the cartilage holds, the styloid will yield, and if it does the continued pull may suffice to tear the internal lateral ligament. (Swett [9])

Sometimes the styloid process is broken off, apparently by avulsion through the lateral ligament or possibly the fibrocartilage. (Stimson [10])

Fracture of the ulnar styloid is probably due to traction of the lateral ligament, pull of the triangular fibrocartilage, or direct violence. (Cotton [3])

The internal lateral ligament attached to the ulnar styloid may be torn off or its angle of pull changed or it may wrench off the end of the styloid process maintaining its own fibres intact. (Speed [11])

The styloid process, intimately attached to the fibrocartilage is fractured by a push of the carpus against the fibrocartilage. (Destot.[12])

The ulnar styloid may be fractured by the internal lateral ligament or the triangular fibrocartilage. In the experimental work of Gallois, the weaker ulnar styloid was found attached to the fibrocartilage. (Schinz [1])

The fracture may be due to either ligament but a complete radial shift of the radial fragment is very unusual so that the internal lateral ligament is probably the causative factor. (Poulsen.[13])

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6. Hennequin, J., and Loewy, R.: Fractures des os, Paris, Masson et Cie, 1904.

7. Duplay, S. E.; Rochard, J. E., and Demoulin, A.: Diagnostic chirurgical, Paris, O. Doin, 1921.

8. Van Lennep, G. A.: Is the Break in the Radius the Only Bone Lesion in Colles's Fracture? Hahneman. Monthly **34**:621, 1899.

9. Swett, P. P.: Colles' Fracture with Special Reference to the Lateral Deformity, New York M. J. **90**:148, 1909.

10. Stimson, L. A.: A Practical Treatise on Fractures and Dislocations, Philadelphia, Lea & Febiger, 1917.

11. Speed, K.: A Text-book of Fractures and Dislocations, Philadelphia, Lea & Febiger, 1928.

12. Destot, Etienne: Traumatismes du poignet et rayons X, Paris, Masson et Cie, 1923.

13. Poulsen, K.: Studien ueber die sogenannte typische Fractur des Radius, Arch. f. klin. Chir. **80**:902, 1906.

It is difficult to tell which ligament produces the fracture, because the ligamentous attachments may vary from the base of the styloid to the tip. (Essau [14])

Direct trauma is hardly likely. Delbet proved that the internal lateral ligament was strong enough to tear off the styloid process. The internal lateral ligament is the most important factor in the fracture. Rochard and Demoulin believe the fibrocartilage to be the causative agent, but the author objects because it would not explain a fracture of the tip. Further if the fibrocartilage inserts at the base of the styloid it would not produce any lesion except a longitudinal line of fracture following the bony canals. This does not occur. (Bouygues.[15])

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14. Essau, P.: Ueber die isolierte Fractur des Processus styloides ulnae, Arch. f. Orthop., Wiesb. **11**:42, 1911.

15. Bouygues, J.: Des fractures isolés de l'extrémité inférieure du cubitus, Faculté de Médecine de Paris, Thése, no. 378, 1904, p. 1.

THE PATHOGENESIS OF THE "STRAWBERRY" GALLBLADDER

(CHOLESTEROSIS OF THE GALLBLADDER) *

ROBERT ELMAN, M.D.
AND
EVARTS A. GRAHAM, M.D.
ST. LOUIS

Moynihan,[1] in 1909, first called attention to small yellowish spots like sand or grit on the mucosa of the gallbladder. In such cases he noticed also that the bile sometimes shimmered because of the presence of cholesterol crystals. In the following year, MacCarty,[2] because of the appearance of the organ in these cases, used the descriptive term of "strawberry" gallbladder. Lichtwitz,[3] in 1914, pictured a beautiful instance of the condition and stated that according to his surgical colleagues it was a not infrequent finding at operation. He was not aware that Moynihan and MacCarty had already described it before him. For a time its existence was generally overlooked, and it is true that such a gallbladder may, from its external appearance, seem quite normal and occasionally on microscopic section may show a minimum of inflammatory change. The gross appearance of the mucosa is striking, however, and in recent years it has become more generally known and is now claimed to comprise about one fifth of all types of diseased gallbladders removed at operation.[4]

Boyd [5] published the first detailed study of the pathology of this remarkable condition. He demonstrated the lipoid nature of the white or yellow deposits that are seen with the naked eye in striking contrast to the dark red background of the rest of the mucosa. By histologic and chemical means he also showed that these lipoid-like deposits were cholesterol, an observation, however, that Lichtwitz [3] was really the first to make. The most recent study of Illingworth [4] added much to the knowledge of this type of gallbladder disease, clinically as well as experimentally.

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* Submitted for publication, July 30, 1931.

* From the Department of Surgery, Washington University School of Medicine, and Barnes Hospital.

1. Moynihan, B. G. A.: A Disease of the Gallbladder Requiring Cholecystectomy, Ann. Surg. **50**:1265, 1909.

2. MacCarty, W. C.: Ann. Surg. **51**:651, 1910.

3. Lichtwitz, L.: Ergebn. d. inn. Med. u. Kinderh. **13**:1, 1914.

4. Illingworth, C. F. W.: Brit. J. Surg. **17**:203, 1929.

5. Boyd, W.: Brit. J. Surg. **10**:337, 1922.

The explanation of these extensive cholesterol deposits has generally been that there was a disturbance in the normal absorbing power of the gallbladder mucosa for this substance. Aschoff [6] was apparently the first to maintain that cholesterol is normally taken up by the wall of the gallbladder from the bile. Boyd [5] adopted this theory, adding a bit of indirect evidence of his own in its support. Illingworth [4] also accepted it, though with some reserve, on the basis of additional experiments. Mentzer,[7] arguing inferentially from his own observations, also believed that cholesterol was absorbed by the gallbladder. This theory seems to have been generally adopted by most surgeons at the present time.

In contrast to these views, evidence has been obtained in a series of experiments in this laboratory that seems to show that: (1) the gallbladder does not absorb cholesterol; (2) it has the power of excreting cholesterol, and (3) inflammation may accelerate this excretion. Its bearing on the problem of the pathogenesis of the "strawberry" gallbladder is obvious.

Some of this evidence in favor of the excretion of cholesterol has been briefly described in preliminary communications from this laboratory,[8] but will be presented in detail later in this article. Additional data will be found together with a complete discussion of the cholesterol function of the gallbladder in another report.[9] Our experimental and clinical evidence may be conveniently summarized as follows:

1. When the gallbladder was isolated by ligature of the cystic duct, the bile therein after a sojourn of from two to sixteen days came to contain a larger amount of cholesterol in all but one of nine experiments (tables 1 and 2). In these eight dogs the gallbladder contents (after corrections due to change in volume through dilution or concentration) showed increases of 520, 54, 88, 77, 18, 49, 400 and 151 per cent in their total cholesterol over that present at the time the cystic duct was tied. In one dog there was a decrease of 14 per cent, an amount that we believe was perhaps within the experimental error. These increases obviously could have come only from the wall of the gallbladder. Torinoumi,[10] whose results have been repeatedly quoted in proof of the absorption theory, had performed several experiments similar to these. In three dogs he found definite decreases in the total cholesterol, but they were not marked. In two dogs he found an increase, as we did, but he stated that the tissue showed inflammatory

6. Aschoff, L.: München. med. Wchnschr. **38**:1847, 1906.

7. Mentzer, S. H.: Am. J. Path. **1**:383, 1925.

8. Elman, R., and Taussig, J. B.: Proc. Soc. Exper. Med. & Biol. **28**:1066, 1931.

9. Elman, R., and Taussig, J. B.: J. Exper. Med. **54**:775, 1931.

10. Torinoumi, K.: Beitr. z. path. Anat. u. z. allg. Path. **72**:456, 1924.

TABLE 1.—*Increases in Cholesterol Concentration of Bile After a Sojourn in the Gallbladder (by Ligature of the Cystic Duct)* *

Dog	Duration of Experiment, Days	Cholesterol of Bile: At Operation, Mg./Cc.	Cholesterol of Bile: At Autopsy, Mg./Cc.	Cholesterol of Bile: Change in Percentage	Concentration of Bile as Shown by Bilirubin Content: At Operation, Mg./Cc.	Concentration of Bile as Shown by Bilirubin Content: At Autopsy, Mg./Cc.	Concentration of Bile as Shown by Bilirubin Content: Change in Percentage	Condition of Gallbladder at Autopsy
25	6	0.42	2.71	+520	0.071	0.071	0	Gallbladder full of thick mucoid bile; no signs of infection in the gross
A	1	0.50	0.88	+ 76	0.041	0.050	+ 22	Gallbladder full of thick bile
B	2	0.75	1.45	+ 93	0.061	0.127	+107	Gallbladder full of thick bile
320	14	0.95	1.20	+ 26	0.92	0.34	— 62	Gallbladder full of thin mucoid bile; section shows no inflammatory changes
324	13	1.15	1.50	+ 31	1.3	0.70	— 46	Gallbladder full of thin mucoid bile; section shows dilated vessels but no leukocytic infiltration
166	15	0.79	....	.....	1.4	....	.....	Gallbladder shrunken and contained colorless débris, which consisted of cholesterol crystals; section showed a thick wall but no inflammation or fibrosis
WH	7	0.42	0.78	+ 86	3.33	1.18	— 65	Gallbladder contained thick mucoid bile; section showed moderate inflammatory changes; mucosa contained much cholesterol

* That this increase was not due to concentration of the bile is shown by the figures on bilirubin, which in three cases show that the bile was actually diluted during its sojourn.

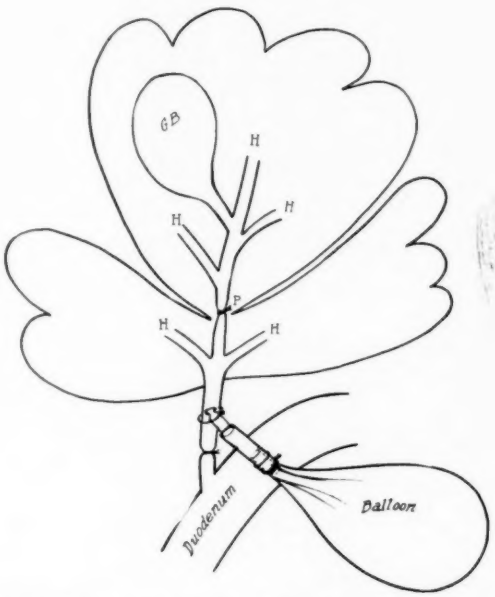
TABLE 2.—*Increase in Total Cholesterol of Bile During its Isolation in the Gallbladder by Ligation of a Cystic Duct* *

Dog	Duration of Experiment, Days	At Operation: Cholesterol, Mg./Cc.	At Operation: Amount of Bile, Cc.	At Operation: Total Cholesterol, Mg.	At Autopsy: Cholesterol, Mg./Cc.	At Autopsy: Amount of Bile, Cc.	At Autopsy: Total Cholesterol, Mg.	Increase in per Cent	Condition of Gallbladder
B45	16	0.36	10	3.6	1.94	8.0	15.52	300	Mucosa showed much cholesterol in section; no signs of inflammatory change
WH	3	0.27	15	4.1	0.76	8.0	6.10	49	Mucosa showed some edema of stroma but no infiltration of leukocytes
830	2	0.50	13	6.5	0.90	8.5	7.65	18	Section showed only edema of wall; localized peritonitis; peritoneal fluid contained 1 mg. per cc. of cholesterol

* The total content was removed and a measured amount reintroduced, leaving only enough for analysis.

changes on section. Infection was present in only a few of our experiments, but we believe that the increase was more marked in them.

2. Experiments designed to compare bile subjected to gallbladder influence with that flowing directly from the liver, in the same dog, showed a much greater concentration of cholesterol in the former even when one corrected for the inspissating effect of the gallbladder.[8] The accompanying sketch shows diagrammatically the nature of these experiments. In one dog the bile in connection with the gallbladder contained 1.1 mg. of cholesterol per cubic centimeter, whereas that coming directly from the liver contained but 0.06 mg. In five other experiments,



Diagrammatic sketch of one series of experiments summarized in the text (see table 3). The partitioning ligature (*P*) separated the bile in two parts. (The gallbladder was first emptied.) The upper part of of the liver drained into the gallbladder (*GB*); the lower part directly into the balloon. *H* indicates the hepatic ducts.

although the bile pigment output by the two parts of the liver drained was equal, the cholesterol output by the part still connected with the gallbladder was 0.38, 0.20, 0.059, 0.077 and 0.105 mg. per gram of liver, whereas the cholesterol output by the rest of the liver was 0.15, 0.09, 0.047, 0.065 and 0.063 mg. per gram of liver, respectively. In other words, the bile made to come in contact with the gallbladder contained thereby 153, 122, 27, 18 and 67 per cent more cholesterol than that which had not. Table 3 summarizes the findings in detail.

TABLE 3.—*Increase in Bile Cholesterol from Contact with the Gallbladder* *

Dog	Duration of Experiment, Hours	Gallbladder Bile						Hepatic Bile						Comment †
		Liver Drained, Gm.	Cholesterol, Mg. per Cc.	Bilirubin, Mg. per Cc.	Amount, Cc.	Bilirubin, Mg. per Gm. of Liver	Cholesterol, Mg. per Gm. of Liver	Cholesterol, Mg. per Gm. of Liver	Bilirubin, Mg. per Gm.	Liver Drained, Gm.	Cholesterol, Mg. per Cc.	Bilirubin, Mg. per Cc.	Amount, Cc.	
Y	48	265	0.72	8.58	14	0.45	0.38	0.15	0.49	172	0.13	4.25	20	Gallbladder section showed acute inflammatory changes; regular diet
P	48	300	0.73	2.86	75	0.72	0.20	0.09	0.71	115	0.074	0.77	140	Gallbladder grossly normal; high cholesterol diet (eggs)
AA	48	51	1.2	12.00	2.5	0.59	0.059	0.047	0.62	192	0.15	2.00	60	Liver pale at operation and autopsy; high cholesterol diet (brains); gallbladder small and shrunken
BB	48	135	0.74	6.67	14	0.69	0.077	0.065	0.49	65	0.19	1.43	22	High cholesterol diet (brains); gallbladder looked normal, and showed no inflammatory changes on section
EE	49	100	0.75	10.5	14	1.61	0.105	0.063	1.47	332	0.097	2.50	215	Gallbladder looked normal and showed no inflammatory changes on section; regular diet

* Comparative cholesterol and bilirubin concentrations of hepatic and gallbladder biles were obtained simultaneously from the same dogs. A partitioning ligature was placed so that part of the bile was diverted to the gallbladder and part was collected in a balloon (see legend of figure). The middle columns show the output of cholesterol per gram of liver drained. The bile from the gallbladder in each case contained much more cholesterol than the other sample.

† Bile was removed from gallbladder at operation by puncture through liver in all cases except dog EE, in which catheter was used and passed up the common duct.

3. Analyses of "white" bile, a product obtained from the isolated bile ducts, showed the presence of cholesterol, which suggests that the entire biliary tract is able to secrete this substance. The colorless contents in human cases of hydrops of the gallbladder also contained cholesterol, a finding that indicates that it had not only not been absorbed with the other biliary constituents but had probably been secreted by the wall of the viscus. Table 4 shows the details of these observations.

TABLE 4.—*Cholesterol Content of the Fluid in Hydrops of the Gallbladder and of the So-Called "White Bile"*

Case	Cholesterol Content, Mg. per Cc.	Description
G. C. C.	0.45	Hydrops of gallbladder removed at operation; many small cholesterol stones in gallbladder and one large one impacted in cystic duct; section of wall showed no inflammation
J. J. E.	0.19	Hydrops of gallbladder removed at operation; wall thick and inflamed; mucous membrane contained cholesterol on section; tiny cholesterol stones adherent to its surface
R. M.	Macroscopic cholesterol crystals	Chronically inflamed gallbladder removed at operation, containing colorless bile
S. J.	Macroscopic cholesterol crystals	Carcinoma obstructing cystic duct; gallbladder full of colorless débris containing cholesterol crystals
Dog Q	0.047	Common duct isolated between ligatures and intubated; 3 cc. collected in rubber balloon during forty-eight hours; sediment contained cholesterol crystals but no bacteria
Dog T	0.085	Same experiment; 4 cc. of clear secretion collected in forty-eight hours; no evidence of infection
Dog HH	0.074	Same experiment; 9.9 cc. of colorless secretion in seventy-two hours; section of common duct showed no inflammation
Dog GG	0.047	Same experiment; 10 cc. collected in ninety-six hours; section showed slight inflammatory changes
Dog 830	0.20	Same experiment; 2 cc. of secretion in forty-eight hours; heavily infected; section showed acute inflammatory signs
Dog 13	0.22	Accidental total biliary obstruction; 5 cc. of infected colorless secretion from dilated hepatic ducts
Dog B44	0.50	Cystic duct tied and gallbladder intubated six days before; 2 cc. of infected secretion collected

4. Comparative analyses [9] of gallbladder and hepatic bile from the same source (human as well as canine) showed that the cholesterol content of the former is much greater than can be accounted for by concentration of the bile. In a series of four dogs, the gallbladders were removed at operation and the common duct drained. The cholesterol concentration of the gallbladder bile was 230, 370, 511 and 490 per cent greater than the average of the hepatic bile collected subsequently in each case. That these figures were not due to inspissation of the bile by the gallbladder was shown by the comparative bilirubin

concentrations;[11] i. e., in the four dogs, respectively, there were —27, +2, +77 and —46 per cent differences in the gallbladder over the hepatic bile. In other words, the bile was concentrated to the extent of but 2 and 77 per cent in two of the dogs, whereas in the other two there was actually a dilution in the gallbladder (presumably due to the secretion of mucus). These specimens were all sterile. In a series of six human beings on whom cholecystectomy was performed similar changes were observed. In three of them the gallbladder bile and hepatic bile were obtained simultaneously, the latter through aspiration of the common duct at operation. In the other three the common duct was drained, and hepatic bile was obtained subsequent to operation. All specimens were filtered to exclude cholesterol-containing cells or débris. The differences in cholesterol content of the gallbladder over the hepatic biles in five of them were + 350, + 470, + 720, + 71 and + 105 per cent, whereas the differences in bilirubin content were + 74, + 44, + 226, + 11 and + 33 per cent, respectively. In other words, the cholesterol content of the gallbladder bile was greater than could be accounted for by concentration of the bile alone. The obvious inference is that the gallbladder mucosa had contributed to this increase.

COMMENT

To explain the development of the "strawberry" gallbladder on the basis of absorption of cholesterol seems unwarranted from the evidence mentioned. It is difficult to see, moreover, how one can explain the storage of cholesterol according to this theory. This substance is found in great quantities in the stroma of the mucosa, and if it had arrived there by absorption through the epithelial cells, what had prevented it from going further? It has been suggested that inflammation may have obstructed the lymphatics, its normal path of absorption. However, proof of this idea is still lacking. Winkenwerder[12] has shown experimentally, for example, that for certain soluble substances the blood vessels and not the lymphatics are the most important pathway of absorption from the gallbladder. Moreover, obstructed lymphatics have not been demonstrated in the "strawberry" gallbladder. Attempts, finally, in this laboratory to produce cholesterosis of the gallbladder by obstructing lymphatics have uniformly been unsuccessful.[13]

In contrast to the inadequacies of this theory, the "strawberry" gallbladder is relatively easy to explain on the basis that cholesterol is

11. Since in these experiments bilirubin is indifferent to secretion or absorption by the biliary mucosa, its measurement furnishes a convenient gage of changes in volume due to the action of the gallbladder on the bile.

12. Winkenwerder, W. L.: Bull. Johns Hopkins Hosp. **46**:272, 1930.

13. Copher, G. H.; Illingworth, C. F. W., and Elman, R.: Unpublished observations.

excreted by its mucosa, particularly in the presence of inflammation. First of all, sufficient evidence has been adduced, it is believed, to indicate that this is a normal phenomenon. The rôle of inflammation in accelerating this excretion is suggested not only by our own experiments, but by the old observation that inflammatory exudates are generally rich in cholesterol.[14] This is of importance, because inflammation of the wall of the "strawberry" gallbladder is usually demonstrable. More cholesterol, moreover, is actually present in the bile in these cases. Illingworth [4] was apparently the first to make this interesting observation, and in a number of our own analyses we also have found very high values, in one instance as much as 9 mg. per cubic centimeter, or over three times the amount usually present in the bile of other gallbladders not showing cholesterosis. The observation of shimmering cholesterol crystals in the bile of these cases by Moynihan has already been referred to.

The storage of cholesterol under the mucosa thus becomes easily explainable whenever the bile for one reason or another becomes unable to take up any more of it. Bile salts play an important rôle in the solution of cholesterol, and it is of interest to note that these two constituents of the bile behave toward the gallbladder in opposite ways. Rosenthal and Licht [15] have recently shown that bile salts are normally absorbed by the gallbladder wall, and that inflammation accelerates this absorption. If this is confirmed, there is, then, an additional factor that operates in the same direction. Thus, it is theoretically possible for stasis alone, by diminishing the bile salt concentration, to lower the solubility of bile for cholesterol, which may then remain in the wall of the gallbladder because the bile can take up no more of it. Infection makes this mechanism more pronounced and is probably necessary to bring about the marked changes found clinically. This is borne out by the experiments of Illingworth,[4] who has reproduced the "strawberry" gallbladder in rabbits on a diet high in fat by infecting the wall of this viscus with streptococci obtained from diseased human gallbladders.

On the other hand, one cannot conclude definitely from these theoretical considerations that stasis is a factor of primary importance in the production of the "strawberry" gallbladder because of other clinical evidence that it is difficult to reconcile with such considerations. This evidence is of two kinds. In the first place, it has been shown by Sherwood Moore [16] that the type of person who is least prone to cholecystic disease in general (and this includes both calculi and the "straw-

14. Wells, H. G.: Chemical Pathology, ed. 5, Philadelphia, W. B. Saunders Company, 1925, p. 300.

15. Rosenthal, F., and Licht, H.: Klin. Wchnschr. **7**:1952, 1928.

16. Moore, Sherwood: Am. J. Roentgenol. **13**:515, 1925.

berry" gallbladder), namely, the asthenic or the splanchnoptotic type, shows on cholecystographic examination to have the slowest emptying time of the gallbladder and therefore the most stasis. The type, on the contrary, that is most prone to have cholecystic disease, namely, the asthenic or hypersthenic person, who is robust, with large muscles, and inclined to obesity, normally shows the fastest emptying time of the gallbladder and therefore has the least stasis. In the second place, it is well known, following the observations of Courvoisier, that in the majority of instances of even prolonged obstruction of the common duct by malignant disease, in which there is complete stasis of the bile in the gallbladder, there is a noteworthy absence of gallstones or of cholecystic disease. From all of this the conclusion seems inevitable that infection is the factor of major importance in the pathogenesis of the "strawberry" gallbladder.

SUMMARY

Experiments have been described and the evidence discussed to show that the gallbladder excretes (rather than absorbs) cholesterol, particularly in the presence of inflammation. An explanation of the pathogenesis of the "strawberry" gallbladder is presented on this basis.

NOTE.—Since this article was submitted for publication the papers of Andrews, Schoenheimer and Hrdina [17] have appeared. These authors also have found that after ligation of the cystic duct there is an increase in the amount of cholesterol in the gallbladder, but they expressed the belief that the increase is due to a differential absorption of bile acids and cholesterol. They apparently did not take into consideration the possibility of the excretion of cholesterol by the mucosa of the organ.

17. Andrews, E.; Schoenheimer, R., and Hrdina, L.: Etiology of Gall Stones: I, II and III, Proc. Soc. Exper. Biol. & Med. **28**:944, 1931.

FRACTURE OF THE SKULL IN CHILDREN*

JAY IRELAND, M.D.
CHICAGO

This report is based on a study of 80 cases of fracture of the skull in patients under 12 years of age admitted to the Children's Memorial Hospital during the years from 1913 to 1930, inclusive. It includes all proved cases of fracture of the skull, the records of which are available, in the charity service during that period. All the cases included were proved to be cases of fracture of the skull by roentgen examination or by roentgen examination and operation at which the fracture was actually demonstrated. All doubtful cases have been excluded from this report because of the great confusion that exists in medical literature regarding the prognosis and treatment in fracture of the skull. It is believed that by basing this study only on proved cases definite conclusions may be drawn. It is probable that some cases of fracture of the skull were in the hospital that are not included in this report, as it has been shown by Apfelbach and LeCount,[1] Troell and Holmström,[2] Kulcke,[3] Moody,[4] Bissell and LeCount,[5] McClure and Crawford[6] and others that a fracture of the skull is often not demonstrable by means of a roentgenogram.

ETIOLOGY

Table 1 gives the sex and age of the patients and the traumatic agents producing the fractures. Of the 80 patients, 54 (67.5 per cent) were boys, and 26 (32.5 per cent) were girls. The youngest patient

* Submitted for publication, April 28, 1931.

* From the Surgical Service of Dr. A. H. Montgomery at the Children's Memorial Hospital.

1. Apfelbach, C. W., and LeCount, E. R.: Pathologic Anatomy of Traumatic Fractures of Cranial Bones and Concomitant Brain Injuries, J. A. M. A. **74**:501 (Feb. 21) 1920.

2. Troell, Abraham, and Holmström, P.: The Importance of Roentgen Examinations in the Diagnosis of Fractures of the Skull, Ann. Surg. **84**:202 (Oct.) 1927.

3. Kulcke: Ein Beitrag zur Frage der Therapie der Schadelbasisfracturen, Deutsche Ztschr. f. Chir. **166**:274 (Sept.) 1921.

4. Moody, W. B.: Traumatic Fracture of the Cranial Bones, J. A. M. A. **74**:511 (Feb. 21) 1920.

5. Bissell, W. W., and LeCount, E. R.: The Relative Frequency of the Various Causes of Coma, J. A. M. A. **68**:500 (Feb. 17) 1917; A Consideration of the Relative Frequency of the Various Forms of Coma, ibid. **64**:1041 (March 27) 1915.

6. McClure, R. D., and Crawford, A. S.: The Management of Craniocerebral Injuries, Arch. Surg. **16**:451 (Feb.) 1928.

was aged 46 days and the oldest 11 years; the average age was 4 years. The groups from 9 to 10 and from 10 to 11 years old had the smallest number of patients injured, there being only 2 patients in each of these groups. The group from 3 to 4 years had the greatest number injured, there being 13 patients in this group. This is the age at which walking is uncertain and the gait unsteady, and at which falls are frequent; consequently, at this age fracture of the skull is common.

Falls were the most frequent causative agent, producing 62.5 per cent of the injuries. Many of the falls were from apparatus on playgrounds. Automobiles caused the injury in 25 per cent of the patients. McClure and Crawford [6] reported that 68.7 per cent of their patients under 10 years of age were injured by automobiles. Despite repeated laws made to control traffic, the automobile continues to be a large

TABLE 1.—*Traumatic Agents According to Age and Sex*

Age, Year	Automobile		Fall		Blow		Street Car		Gunshot		Struck by Bicycle		Crushed		Not Stated		Total		Both Sexes
	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	M	F	
46 days to 1 year	..	..	1	1	..	..	1	..	..	..	..	..	..	..	..	..	2	1	3
1 to 2	..	..	5	3	..	..	..	..	1	..	..	..	..	..	..	..	6	3	9
2 to 3	2	..	5	4	..	..	1	..	..	..	..	..	..	..	..	..	8	4	12
3 to 4	3	..	6	4	..	..	..	..	..	..	..	..	..	..	..	..	9	4	13
4 to 5	2	1	2	..	..	1	..	1	..	..	..	..	..	..	..	..	4	3	7
5 to 6	1	..	4	..	..	..	..	..	..	..	1	..	..	..	..	..	6	0	6
6 to 7	1	2	5	1	..	..	..	..	..	..	..	..	..	1	..	..	6	4	10
7 to 8	3	1	3	..	..	..	..	..	..	..	..	..	..	..	1	..	7	1	8
8 to 9	..	3	3	1	1	..	..	..	..	..	..	..	..	..	..	..	4	4	8
9 to 10	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	1	1	1	2
10 to 11	1	..	..	1	..	..	..	..	..	..	..	..	..	..	..	..	1	1	2
Totals	13	7	35	15	1	1	2	1	1	..	1	..	..	1	1	1	54	26	80
Totals, both sexes	20		50		2		3		1		1		1		2		80		
Percentage of 80 cases	25		62.5		2.5		3.7		1.2		1.2		1.2		2.5		100		

factor in producing fracture of the skull. Children at the unsteady age of from 3 to 4 years, as noted in the foregoing paragraph, often become separated from the parent or attendant accompanying them on the street and walk into the path of a passing automobile, and a fracture of the skull is the result. Among the more infrequent causes were blows, street car and bicycle accidents, crushing injuries and gunshots.

LOCATION AND EXTENT OF FRACTURES

Table 2 classifies the fractures as to whether they were linear and as to whether they were depressed or elevated; also as to what bone or bones were fractured. The parietal bone was fractured more frequently than any other. In this series 45 patients showed a fracture of the parietal bone, 17 of this bone alone and 28 of the parietal bone and of other bones of the skull. Fracture of the frontal bone was next in frequency; there were 35 patients in this group, 17 with a

fracture of the frontal bone only and 18 with a fracture of the frontal bone and of other bones of the skull. No patient with a fracture of the frontal bone acquired meningitis or abscess of the brain, although this is frequently reported subsequent to a fracture extending into the frontal sinus. Eight patients had depressed fractures. One of these fractures was not visible in the roentgenogram, but was found at operation. Three patients had definite elevation of the fragment, a condition rarely described in the literature. None of them was operated on, and all recovered. Sixty-three patients (78.7 per cent) had a fracture of the vault alone, 8 (10 per cent) of the base alone and 8 (10 per cent) of both vault and base; the location of the fracture in 1 patient (1.2 per cent) was not stated. Only 2 patients (2.5 per cent) had separation of the sutures, a condition frequently described in the literature on fracture of the skull in children. It would seem that the percentage

TABLE 2.—*Classification of Fractures*

	Linears			Depressed					
Bone Fractured	Right Side	Left Side	Side Not Stated	Right Side	Left Side	Side Not Stated	Elevated	Bilateral	Total
Frontal	12	7	1	1	1	1	..	1	23
Parietal	6	4	5	1	1	1	2	..	18
Occipital	..	..	8	..	..	..	..	..	8
Temporal	1	1	..	..	..	..	..	..	2
Frontoparietal	3	5	1	..	..	1	..	1	11
Frontotemporal	..	..	1	..	..	..	..	..	1
Parieto-occipital	1	4	3	..	..	..	..	..	8
Parietotemporal	1	3	1	..	..	1	..	..	6
Fronto-ethmoid	1	..	..	..	..	..	..	..	1
Tempero-occipital	2	..	..	..	..	..	..	..	2
Frontoparieto-occipital	2	2	..	..	..	..	..	1	5
Temporoparieto-occipital	..	..	..	1	..	..	..	..	1
Frontoparietotemporal	..	..	..	..	..	..	1	..	1

of basal fractures in children is not so high as that of basal fractures in adults. Many of the basal fractures in adults are fractures by contrecoup, but it is doubtful whether the percentage of contrecoup fractures in children is as high as it is in adults. Hence, the percentage of basal fractures in children would be less. Among the various investigators who have reported series in adults, Apfelbach and LeCount [1] found fracture of both vault and base in 92 per cent of their patients; Besley,[7] in 34 per cent. The average age of 68.7 per cent of Besley's patients was over 30 years. Stewart [8] reported basal fractures in 34 per cent and fractures of the vault in 66 per cent of his patients, the ages of whom averaged 36 years. On the other hand, Beekman [9] reported that 74

7. Besley, F. A.: A Contribution to the Subject of Skull Fractures, J. A. M. A. **66**:345 (Jan. 29) 1916.

8. Stewart, J. W.: Fractures of the Skull, J. A. M. A. **77**:2030 (Dec. 24) 1921.

9. Beekman, Fenwick: Head Injuries in Children, Ann. Surg. **87**:355 (March) 1928.

per cent of his patients, all of whom were under 13 years of age, had basal fractures.

The lengths of the fractures in 58 patients were measured as seen in roentgenograms. The longest fracture, which completely encircled the vault, was 36 cm.; the shortest was 0.6 cm.; the average length was 8.4 cm. Eleven patients were examined roentgenographically a second time. The shortest period of time between the first and the second examination was 282 days, the longest period was 6 years and 12 days, and the average interval was 647 days. In every patient except 1, the fracture had completely closed at the second examination, and there was no evidence of thickening or of callus. In the patient whose fracture had not completely healed, as shown by the roentgenogram, the fracture extended entirely around the vault, as noted, and was plainly visible 610 days after the injury. Stewart [10] stated that there is little formation of callus following fracture of the skull, and that all roentgenographic evidence of fracture of the skull in a child may disappear within from 6 to 8 months. He also stated that in an adult a simple linear fracture may remain visible for 3 years, and that if there is much separation, the defect may remain visible for from 3 to 5 years.

In the cases analyzed here, there seemed to be no more tendency for a fracture of the vault to extend into the base than to extend in any other direction. This is the opposite of what is true for adults, in whom the fracture has a great tendency to follow between the heavy arches into the base, as described by Apfelbach and LeCount.[1]

SYMPTOMS

Many of these patients arrived at the hospital in police ambulances only a few minutes after they had been injured. Sixty-six (82.5 per cent) entered the hospital on the same day that they were injured. The remaining 14 (17.5 per cent) entered from 1 to 100 days after injury.

Thirty-two patients (40 per cent) were unconscious immediately following the accident, and 12 (15 per cent) were unconscious on entrance into the hospital. Eighteen (22.5 per cent) regained consciousness between the time of injury and the time of entrance into the hospital.

Sixty-eight (85 per cent) had a visible laceration, hematoma or contusion of the head. Headache was present in 17 patients (21.2 per cent), dizziness in 9 (11.2 per cent) and vomiting in 51 (63.7 per cent), with bloody vomitus in 18 (22.5 per cent). From these percentages, vomiting would seem to be an important symptom of fracture

10. Stewart, W. H.: The Time Factor in the Disappearance of Roentgenographic Evidence of Fractures of the Skull, Brit. J. Radiol. **30**:399 (Nov.) 1925.

of the skull in children. When vomiting follows an injury to the head, one should immediately suspect that there is a fracture of the skull.

In 66 patients (82.5 per cent) the pupils were equal, and in 11 (13.7 per cent) they were unequal. The right pupil was larger in 6 patients; the left, in 4, and in 1 it was not stated which was larger. In 2 patients (2.5 per cent) both pupils were fixed and dilated; these patients died. Phelps [11] reported that in patients with fixed and dilated pupils the mortality was 100 per cent. Stewart [8] also noted a mortality of 100 per cent in 53 patients with fixed and dilated pupils. In the present series there were 73 patients (91.2 per cent) in whom the pupils reacted to light, and 4 (5 per cent) in whom they did not; the pupillary reactions of 3 patients were not stated. In most of the patients, because of their age, the test for the reaction to accommodation was unsatisfactory.

Examination of the eyegrounds was not considered of much importance, except in patients whose severe symptoms lasted several days. The fundi were examined in 6 patients. Three had normal fundi; 1 had a hazy disk, 1 had definite papilledema and 1 had a retinal hemorrhage. All recovered. Jackson [12] intimated that examination of the eyegrounds is of little value in fracture of the skull. Moorhead and Weller [13] stated that the importance of choked disk is much overrated. Speed [14] found that examination of the eyegrounds was of little prognostic value in early cases. Johnson [15] examined the fundi in 154 patients with cranial and intracranial injuries and noted changes only in 4. In contrast, Cushing [16] thinks that examination of the eyegrounds is of much value, and Shere,[17] Maes (quoted by Crawford [18]) and Haines [19]

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11. Phelps, Charles: An Analytical and Statistical Review of 1,000 Cases of Head Injury, Ann. Surg. **49**:511 (April 1) 1909.

12. Jackson, Harry: The Management of Acute Cranial Injuries by the Early Exact Determination of Intracranial Pressure and Its Relief by Lumbar Drainage, Surg., Gynec. & Obst. **34**:494 (April) 1922.

13. Moorhead, J. J., and Weller, Walter: Fractures of the Skull in Children, Ann. Surg. **74**:72 (July) 1921.

14. Speed, Kellogg: Gunshot Fractures of the Skull, J. A. M. A. **68**:1299 (May 5) 1917.

15. Johnson, J. G. W.: Cranial and Intracranial Injuries, Internat. Clin. **2**:266 (June) 1926.

16. Cushing, Harvey: Subtemporal Decompression Operations for the Intracranial Complications Associated with Bursting Fractures of the Skull, Ann. Surg. **47**:641 (May) 1908.

17. Shere, O. M.: Indications for Operative Treatment in Cranial Fractures, Colorado Med. **17**:9, 1920.

18. Crawford, L. B.: Some Fractures of the Base Treated by Repeated Spinal Punctures, New Orleans M. & S. J. **74**:374, 1921-1922.

19. Haines, W. D.: Fractures of the Skull, Ohio State M. J. **15**:281 (May) 1918.

think likewise. Eagleton [20] said that such an examination may show moderate increase of the intracranial pressure in fracture of the skull when no other symptoms are present, but he had never seen a typical choked disk in a patient with fracture of the skull.

Only 2 patients had paralysis of the extrinsic muscles of the eyes, three had nystagmus, and one had diplopia.

Nineteen patients (23.7 per cent) had bleeding from the nose. Two of these had only a basal fracture, 14 had a fracture only of the vault, and 3 had a fracture of both the base and the vault. It is significant that fracture of the vault should cause such a high incidence of nasal hemorrhage. Twelve patients (63.5 per cent) of the 19 who had nasal hemorrhage had a fracture of the frontal bone. Hence, nasal hemorrhage following an injury to the head should cause one to suspect that there is a fracture of the frontal bone. Eleven patients (14 per cent) had bleeding from the ears, 9 from the left ear alone, none from the right alone and 2 from both ears. One of these had a basal fracture, 7 a fracture of the vault, and 2 a fracture of both the base and the vault. The nature of the fracture in 1 was not stated. In 5 of the patients who had bleeding from the left ear, the left side of the skull was fractured; in 2 who had bleeding from the right ear, and in 3 who had bleeding from the left ear, the right side was fractured. The location of the fracture in 3 patients with bleeding from the left ear was not stated. From the statistics of aural hemorrhage no conclusion can be drawn as regards the bone fractured. Fourteen patients (17.5 per cent) had bleeding from the mouth; 3 of these had a fracture of the base, 9 of the vault and 2 of both the base and the vault.

The blood pressure was recorded 45 times for 22 patients. The highest systolic blood pressure was 152 mm. of mercury, the lowest 80 and the average 106. The highest diastolic pressure was 92 mm. of mercury, the lowest 7 and the average 59. The systolic pressure of one of the patients who died was 96 and the diastolic 70. The blood pressure of the other patient who died was not recorded. The readings of the blood pressure of patients whose pressure was taken more than once tended to be lower the longer the patient remained under observation. It would seem that in accordance with the conclusion of Vance,[21] no definite prognostic conclusion could be drawn from the blood pressure of these patients.

The muscle reflexes were recorded for 71 patients (88.7 per cent). In 4 patients all the reflexes were increased. All reflexes were increased on the right side twice, and the right knee reflex was increased twice. The reflexes were absent on the left side once. The right knee jerk was

20. Eagleton, W. P.: Fracture of the Skull, Arch. Surg. **3**:140 (July) 1921.
21. Vance, B. M.: Fractures of the Skull, Arch. Surg. **14**:1023 (May) 1927.

absent twice and sluggish once; the left knee jerk was absent once. The abdominal reflexes were recorded as absent twice. Babinski's sign was positive 3 times on the right and 3 times bilaterally. Kernig's sign was positive once on the right. All reflexes were absent in 2 patients (one of these died; the other recovered). There was a total of 12 patients (15 per cent) with abnormal reflexes and 68 (85 per cent) with normal reflexes. These statistics indicate that normal reflexes frequently accompany fracture of the skull in children.

The highest temperature of any patient was 105 F., taken rectally; the lowest was 96 F. The lowest average temperature attained by all patients was 95.2 F. (obtained by averaging the lowest temperatures attained by the patients individually). The highest average temperature was 101.4 F., taken rectally (obtained by averaging the highest temperatures attained by the patients individually). The general average temperature was 100.1 F., taken rectally. The lowest rectal temperatures attained by the patients individually in the first 24 hours after admission were averaged, and the average was found to be 98.8 F. There were 8 patients whose temperatures did not fall below 99.6 F. rectally in the first twenty-four hours after admission. This is at variance with the observations of Hipsley,[22] who stated that the temperature is always subnormal in fracture of the skull. It also shows that not all the patients are in a state of severe shock on admission. In Besley's [7] fatal cases the average temperature was 102 F. Courtney [23] held that the prognosis is bad if the temperature is over 102 F., and that the temperature is the most important sign in intracranial traumatism. Eight patients of the series had temperatures over 102 F., taken by mouth (103 F., rectally); seven of these recovered. No definite prognostic conclusions can be drawn from the temperature in these cases, unless it is that cases marked by high temperatures (over 102 F., taken rectally) are not usually fatal in children, as they often are in adults.

In this series, if the patient was perfectly conscious and apparently normal except for the fracture, it was not deemed necessary to record the pulse and the temperature every 15 minutes, as is frequently recommended. The highest pulse rate recorded was 180 per minute, the lowest was 50, and both of the patients recovered. The highest average pulse rate (obtained by averaging the highest pulse rates attained by the patients individually) was 133. The lowest average pulse rate was 79. The general average pulse rate was 106. Regarding the pulse rates of the two patients who died, the pulse of one was constantly imper-

22. Hipsley, P. L.: Fracture of the Skull in Children, M. J. Australia **1**:5 (Jan. 3) 1925.

23. Courtney, J. W.: The Temperature as a Valuable Guide to Diagnosis, Prognosis and Surgical Treatment in Cranio-Cerebral Traumatisms, Boston M. & S. J. **177**:511, 1917.

ceptible, and the pulse rate of the other was not recorded. Twelve patients (15 per cent) had a pulse rate of over 150, and all of these recovered. Hence, a high pulse rate (over 150) would not seem to be a bad sign in fracture of the skull in children. As stated previously, the lowest pulse rate recorded was 50; the pulse rate in fracture of the skull in adults often is much lower than this.

The highest respiration rate attained by any patient was 60, the lowest 15 per minute. The highest average respiration rate (obtained by averaging the highest respiration rates attained by the patients individually) was 34; the lowest average was 19. The general average was 26.5.

COMPLICATIONS

Thirteen patients had fracture of one or more bones in addition to fracture of the skull. The other bones fractured with the number of cases, were: femur, 5; humerus, 2; maxilla, 1; palatinum, 1; forearm, 1; clavicle, 1; tibia, 1, and metatarsal, 1. Also, there were other complications, as follows: tearing of the spinal nerve roots of the brachial plexus, 1; pneumonia, 1; pleurisy, 1; infected wounds, 2, and otitis media, 3. The symptoms that developed as a direct result of the fracture of the skull, with the number of cases, were: flaccid paralysis of the right side of the body, 2; weakness of the right arm and the right leg, 1; deviation of the tongue to the right, 1; generalized convulsions, 1; twitching of the hand, 1; twitchings of facial muscles, 3; twitching of muscles of the arm and leg, 1, and twitching of the eye, 1. No patient died from a complication or from a concomitant injury.

DIAGNOSIS

Lumbar puncture was performed 14 times on 13 patients. The pressure of the spinal fluid was recorded as increased 10 times and as normal twice; in 2 instances the amount of pressure was not stated. The cerebrospinal fluid was bloody 9 times and clear 3 times; in 2 cases the character of the fluid was not stated.

Stewart,[8] Hipsley,[22] Boyd,[24] Eagleton,[20] Shere,[17] Carter,[25] Moorhead and Weller[13] and Haines[19] are in favor of lumbar puncture for diagnosis, while Sachs[26] opposes it. Cushing[27] stated that he was in

24. Boyd, William: The Cerebrospinal Fluid, New York, The Macmillan Company, 1920, p. 33.

25. Carter, B. N.: Diagnosis and Treatment of Fractures of the Skull as Developed in the Cincinnati General Hospital, Ann. Surg. **83**:182 (Feb.) 1926.

26. Sachs, Ernest: Diagnosis and Treatment of Head Injuries, J. A. M. A. **81**:2159 (Dec. 29) 1923.

27. Cushing, Harvey: Some Experimental and Clinical Observations Concerning States of Increased Intracranial Tension, Am. J. M. Sc. **124**:374 (Sept.) 1902.

doubt as to how much stress to place on lumbar puncture for diagnosis. Schoenbeck [28] collected 71 cases in which death occurred following lumbar drainage; in 69 of these at necropsy a tumor or other acute or chronic disease of the brain was found. Hence, unless a history of symptoms of a disease of the brain antedating the injury to the head has been obtained, lumbar puncture seems to be almost without danger. However, as correctly pointed out by Besley,[7] there is sufficient danger so that puncture should not be done if a diagnosis can be made by physical examination, roentgen examination and other means. Little use was made of lumbar puncture in this series for the reason stated, and the diagnosis could usually readily be made without it.

There was no case in which the diagnosis of epidural hemorrhage was made. Vance [21] reported 2 cases of epidural hemorrhage, occurring in patients between 10 and 20 years. Epidural hemorrhage is less frequent in children than in adults, because the dura is more adherent to the skull in children, while in adults it is loosely adherent and allows a clot to form readily between it and the skull. There was no case with a diagnosis of pneumocephalus, as described by Rand.[29] One patient had a fracture of the frontal sinus, as described by Teachenor;[30] infection of the brain did not develop, and the child recovered.

PROGNOSIS

In cases of fracture of the skull the prognosis is much better for children than for adults, for the following reasons: 1. The shock of a surgical operation is less in younger people than in older ones (Brophy [31]). 2. The edges of the bones of the vault of the skull in childhood are connected by a membrane and are comparatively movable, and for this reason the blood vessels that lie directly beneath the fracture are not so readily torn and the brain is not so seriously injured (Piersol [32]). 3. The furrows of the skull in which the blood vessels lie are not so deep in children. 4. Bones in children are more elastic, and there is more tendency toward greenstick fracture. 5. The sutures are less dense and therefore interrupt the transmission of violence to a greater

28. Schoenbeck, O.: Die Gefahren der Lumbalpunktion, Arch. f. klin. Chir. **107**:309, 1915-1916.

29. Rand, C. W.: Traumatic Pneumocephalus, Arch. Surg. **20**:934 (June) 1930.

30. Teachenor, F. R.: Intracranial Complications of Fracture of Skull Involving Frontal Sinus, J. A. M. A. **88**:987 (March 26) 1927.

31. Brophy, W. T.: Oral Surgery, Philadelphia, P. Blakiston's Son & Company, 1915, p. 609.

32. Piersol, G. A.: Human Anatomy, ed. 9, Philadelphia, J. B. Lippincott Company, 1930.

degree. 6. Basal fracture is less common because the heavy arches [33] at the base of the skull are not so pronounced in children, hence, the fracture is not so readily radiated into the base, and because the vertebral column in children is less rigid and does not transmit force to the skull so violently in cases of vertebral injury. 7. Hipsley [22] said that the associated injury to the brain in children is less because the subarachnoid cavity is dilated and acts as a water cushion between the anterior and middle fossae and between the middle and posterior fossae. Levinson,[34] however, stated that there is less cerebrospinal fluid during the first twenty-four hours in the new-born child than in older children. 8. As children have a lower cerebrospinal fluid pressure than adults, the brain is better protected against the force producing the fracture.

Two (2.5 per cent) of the 80 patients died. This mortality is extremely low, as is shown in a comparison of the following statistics: Carter [25] reported 41 cases occurring in patients between the ages of 1 and 10, in which there was a mortality of 14.6 per cent, and 29 cases occurring in patients between the ages of 10 and 20, in which the mortality was 13.7 per cent. Harris and Nissen [35] reported 56 cases in patients aged from 1 to 10, with 24 deaths (42.8 per cent). Twenty-two of these patients were operated on; 7 died (31.8 per cent); of 34 not operated on, 17 lived and 17 died (a mortality of 50 per cent). Johnson [15] reported 20 cases in patients aged from 1 to 10, 3 of whom died (15 per cent). Rand and Nielsen [36] reported 12 cases in patients aged from 1 to 10, with 1 death (8.3 per cent), and 22 cases in patients aged from 10 to 20, with 5 deaths (22.7 per cent). Hipsley [22] reported 27 cases in patients aged from 1 week to 12 years, with 2 deaths (7.4 per cent). Beekman [9] reported a mortality of 13 per cent in 214 cases of fracture of the skull in children under 13 years of age. Moorhead and Weller [13] reported that in 100 cases in children aged from 3 months to 16 the total mortality was 26 per cent. In 12 patients that were operated on the mortality was 33.3 per cent. Mixter [37] reported a mortality of 54.1 per cent in 301 patients, including both children and adults. Brown and Strecker [38] reported 100 cases in children and adults,

33. LeCount, E. R.: The Pathology of Skull Fractures, Northwest Med. **18:** 205, 1919; Apfelbach and LeCount (footnote 1).

34. Levinson, A.: Cerebrospinal Fluid in Infants and in Children, Am. J. Dis. Child. **36:**798 (Oct.) 1928.

35. Harris, C. T., and Nissen, H. A.: A Series of Fractured Skulls from the Surgical Records of the Boston City Hospital 1902-1917, Boston M. & S. J. **177:** 870 (Dec. 20) 1917.

36. Rand, C. W., and Nielsen, J. M.: Fractures of the Skull, Arch. Surg. **2:**434 (Sept.) 1925.

37. Mixter, W. J.: Fractures of the Base of the Skull at Massachusetts General Hospital, Boston M. & S. J. **177:**518 (Oct. 11) 1917.

38. Brown, H. P., Jr., and Strecker, E. A.: Some Observations on the Treatment of Fracture of the Skull, Ann. Surg. **79:**198 (Feb.) 1924.

with 26 deaths (26 per cent); of 17 patients operated on, 6 died, and of 83 not operated on, 20 died. McCreery and Berry [39] reported 33 deaths in 62 cases in adults that were operated on (53 per cent mortality). They had a total mortality of 39 per cent in 204 cases. Shere [17] reported that 67 per cent of his patients (ages not stated) were operated on, with a mortality of 48.8 per cent, and that 33 per cent were not operated on, with a mortality of 59 per cent. Selberg [40] stated that the mortality in fracture of the skull (ages of patients not stated) approaches 50 per cent. Crawford [18] reported 10 patients (ages not stated) treated by lumbar puncture; all recovered.

The prognosis is bad when the pupils are fixed and dilated and when all the reflexes are absent. A high temperature (i. e., over 102 F.), unless it is continued for a long period of time, does not seem to be a bad prognostic sign in children. As noted, 7 of the patients who had temperatures over 102 F. recovered. The injury to the brain is much more important than the fracture of the skull. Many patients die from injuries of the brain in cases in which no fractures can be demonstrated at necropsy. Hence, the prognosis depends chiefly on the extent of the pathologic process in the brain.

TREATMENT

Since Weed and McKibbin [41] showed that the bulk of the brain and the pressure of the cerebrospinal fluid can be reduced by intravenous injection of hypertonic salt solution, and since Foley [42] showed that the flow of cerebropsinal fluid through the choroid plexus is reversed in direction by intravenous or intraduodenal administration of salt, these methods have been extensively used in fracture of the skull.

Hypertonic salt solution given intravenously, as described by Weed and McKibbin,[41] was recommended by Newell,[43] Johnson,[15] Keegan [44] and Grant.[45] On the other hand, Rand and Nielsen [36] and Sachs [26]

39. McCreery, J. A., and Berry, F. B.: A Study of 520 Cases of Fracture of the Skull, Ann. Surg. **88**:890 (Nov.) 1928.

40. Selberg, F.: Nachuntersuchungen an Schadelbrüchen, Deutsche med. Wchnschr. **54**:2099 (Dec. 14) 1928.

41. Weed, L. H., and McKibbin, P. S.: Pressure Changes in Cerebrospinal Fluid Following Intravenous Injections of Solutions of Various Concentrations, Am. J. Physiol. **48**:512 (May) 1919; Experimental Alteration of Brain Bulk, ibid. **48**:531 (May) 1919.

42. Foley, F. E. B.: Alteration in the Currents and Absorption of Cerebrospinal Fluid Following Salt Administration, Arch. Surg. **6**:587 (March) 1923.

43. Newell, E. T.: Diagnosis and Treatment of Fractures of the Skull, J. South Carolina M. A. **25**:461 (July) 1929.

44. Keegan, J. J.: When to Operate and When Not to Operate in Fractures of the Skull, Nebraska M. J. **10**:333 (Sept.) 1925.

45. Grant, F. C.: Chronic Subdural Hematoma, Ann. Surg. **86**:485 (Oct.) 1927.

said that they were not in favor of hypertonic salt solution given intravenously. Haines [19] stated that hypertonic salt solution should not be injected during the period of shock, because it increases the blood pressure. The intravenous injection of dextrose was recommended by Newell.[43] Johnson [15] and Rand and Nielsen [36] were in favor of magnesium sulphate, given either as an enema or orally. Opposing this view, Connors [46] saw no benefit from 50 per cent magnesium sulphate given as an enema.

Lumbar puncture was done on 13 patients—whether for diagnostic or therapeutic purposes was not definitely stated. One patient that had a lumbar puncture died, the puncture being made while the patient was in a state of extreme shock, when it should not have been done, as pointed out by Munroe [47] in relation to extreme shock in the new-born infant. Phelps,[11] Carcassonne,[48] Harris and Nissen,[35] Elder,[49] Newell,[43] Bowen and Van Schaick,[50] Rand and Nielsen,[36] Hipsley,[22] Jackson,[12] Stewart,[8] Boyd,[24] Munroe,[47] Eagleton,[20] Keegan,[44] Crawford,[18] Ferry [51] and Johnson [15] favored lumbar puncture in treatment. Some depend on accurate measurement of the pressure of the spinal fluid to determine the amount of fluid to withdraw and others merely estimate it. There seems to be no uniform rule.

There is a marked disagreement as to what constitutes normal pressure of the cerebrospinal fluid as measured at lumbar puncture. Waitz [52] held that in the new-born infant it is zero; Munroe [47] placed it at from 2 to 6 mm. of mercury, Sidbury [53] at from 2 to 5 mm. and Levinson [34] at from 1 to 6 mm. Quincke [54] found the normal pressure

46. Connors, J. F.: The Treatment of Skull Fractures, Tr. Am. S. A. **45**:426, 1927.

47. Munroe, Donald: Cerebrospinal Fluid Pressure in the New-Born, J. A. M. A. **90**:1688 (May 26) 1928.

48. Carcassonne: Fracture de la base du crâne traitée par ponction lombaire, Lyon méd. **148**:715, 1929.

49. Elder, H. M.: Fracture of the Skull of a Child Treated by Repeated Puncture of the Cisterna Magna, Canad. M. A. J. **15**:1248, 1925.

50. Bowen, Frederick; and Van Schaick, Harold D.: Spinal Puncture as Treatment in Cranial Fractures, J. Florida M. A. **12**:310 (May) 1926.

51. Ferry, G.: Contribution à l'étude du diagnostic et du traitement des fractures de la base du crâne, Rev. de chir., Paris **61**:117, 1923.

52. Waitz, R.: Le liquide céphalorachidien du nouveau-né, Rev. franç. de pédiat. **4**:1, 1928.

53. Sidbury, J. B.: The Importance of Lumbar Puncture in Intracranial Hemorrhage of the New-Born, Arch. Pediat. **37**:545 (Sept.) 1920.

54. Quincke, H.: Ueber den Druck in Transsudaten, Deutsches Arch. f. klin. Med. **21**:453, 1887; Ueber die therapeutischen Leistungen der Lumbalpunktion, Therap. Monatsh. **28**:469 (July) 1914; Die diagnostische und therapeutische Bedeutung der Lumbalpunktion, Deutsche med. Wchnschr. **31**:1825 (Nov. 16) 1905.

in children to be from 40 to 60 mm. of water. Falkenheim and Naunyn [55] found it to be from 50 to 500 mm. of water in children aged from 1 month to 1 year. Levinson [56] found it to be from 45 to 90 mm. of water in children lying and from 150 to 230 mm. in children sitting. Phelps [11] stated that the normal pressure (age not stated) is from 50 to 100 mm. of water. Boyd [24] gave the normal pressure as from 90 to 150 mm. of water (age not stated), and stated further that even if the medulla is forced into the foramen magnum, there may be no increase in the spinal pressure even with high intracranial pressure. Howe [57] stated that the intracranial pressure is roughly equal to the venous pressure. Levinson [58] stated that if more than 10 cc. of fluid can be removed at a single sitting, or if there is a flow of over 10 drops per minute, it indicates an increase in the amount of pressure. Munroe [47] removed as much as 90 cc. of fluid in one case. He stated that the spinal pressure in new-born infants who lived averaged the same as that in those who died; but that in adults, the pressure was 2½ times greater in those whose fractures were fatal than in those whose fractures were not. Cushing [59] stated that the spinal pressure should equal the venous pressure, or about 4 mm. of mercury (no reference was made to age).

Connors,[46] Dandy [60] and Sachs [26] did not favor lumbar puncture in the treatment for fracture of the skull. They expressed the belief that it may do harm and that injury to the base of the brain or even sudden death may result from it. Eagleton,[20] McClure and Crawford,[6] Elder [49] and Johnson [15] favored cysternal or ventricular puncture in some cases.

Therapeutic lumbar puncture should not be done as a matter of routine in cases of fracture of the skull in children. For example, if a patient is perfectly conscious, has no evidence of intracranial pressure, as shown by the pulse, the blood pressure, the respiration or other physical signs, puncture should not be made. With signs of increasing pressure, it may be carefully done, the fluid being drained off very slowly and the drainage being discontinued if any untoward symptoms develop.

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55. Falkenheim, H., and Naunyn, B.: Ueber Hirndruck, Arch. f. exper. Path. u. Pharmakol. **22**:261, 1887.

56. Levinson, Abraham: Cerebrospinal Fluid, ed. 3, St. Louis, C. V. Mosby Company, 1929.

57. Howe, H. S.: Physiologic Mechanism for the Maintenance of Intracranial Pressure, Arch. Neurol. & Psychiat. **20**:1048 (Nov.) 1928.

58. Levinson, Abraham: Cerebrospinal Fluid, in Abt, I. A.: Pediatrics, Philadelphia, W. B. Saunders Company, 1923, vol. 2, p. 96.

59. Cushing, Harvey: Some Experimental and Clinical Observations Concerning States of Increased Intracranial Tension, Am. J. M. Sc. **124**:376 (Sept.) 1902.

60. Dandy, Walter: Injuries to the Head, J. M. Soc. New Jersey **27**:91 (Feb.) 1930.

If tapping the spinal fluid does not relieve the symptoms of pressure, surgical decompression may be done. Surgical decompression was done in 7 patients (8.7 per cent), 1 of whom died. Two were operated on for depressed fractures; the depression was elevated in the one, and bony fragments were removed in the other. Seven patients (8.7 per cent) had depressed fractures on whom operation was not done. All of these recovered. This is in accord with Beekman,[9] who did not think it necessary to operate on all patients with depressed fractures. The 4 patients with elevated fractures were not operated on, and all recovered. Two patients were operated on who had no depression. One was operated on for removal of a bullet; this patient recovered. One patient operated on was found to have no pathologic condition of the brain; this patient died.

Wagner [61] in 1889 first introduced the flap of bone and soft tissue in operations on the skull: the omega-shaped flap is turned down and replaced at the close of the operation. It leaves little defect. Cushing's [16] subtemporal decompression tends to prevent hernia cerebri, because it is beneath the temporal muscle. Jackson [12] considered that the decompression advised by Cushing does not adequately relieve the pressure on the medulla, probably because the pressure is subtentorial and the operation supratentorial. Vance [21] found the pons actually flattened by being pushed against the basilar process when both subdural spaces were distended with extravasated blood, which shows that pressure above the tentorium may be transmitted to the base of the brain. It is logical that if most of the injury is above the tentorium, most of the edema of the brain will be there; hence the decompression above the tentorium would be most beneficial. Cerebellar injuries (i. e., those below the tentorium) are not so common as those above the tentorium (i. e., cerebral). There is at times a question as to the actual amount of edema of the brain. In the 512 necropsies of Vance [21] no death was found to have resulted from edema of the brain. He did not find the marked swelling of the brain that LeCount and Apfelbach found. Courtney [23] stated that trephining for the relief of edematous compression in adults is for the most part a fruitless expedient. Perkins (quoted by Crawford [18]) stated that the end-results of decompression are poor, and that paralysis and imbecility may occur.

Pollak [62] thought that comminuted fractures of the vault should have a thorough exploration. Newell [43] considered a pulse rate of 50 or less sufficient indication for operation. Eagleton [20] stated that a diastolic pressure below 55 is a contraindication for operation because

61. Wagner, W.: Die temporäre Resektion des Schadeldachs an Stelle der Trepanation, Zentralbl. f. Chir. **16**:833 (Nov. 23) 1889.

62. Pollak, A. W.: Unrecognized Fractures of the Skull in Infants and Children, M. J. & Rec. **119**:65 (April 2) 1924.

the patient is in a state of shock. Keegan [44] and McCreery and Berry [39] thought that a patient suffering from shock should not be operated on. In this group the patient who died was operated on while in this condition. This, beyond doubt, contributed to the fatality.

Mixter [37] said that he removes the loose and depressed fragments of bone. Speed [14] said that in gunshot fractures he removes the loose fragments of bone. On the other hand, Cushing [63] said that he is inclined to replace the fragments; however, he did not think defects left in the skull are injurious unless accompanied by an underlying lesion of the dura. Hanson and his associates [64] held that moderate-sized defects left in the skull by operation should be closed, and he reported good results from closing the defect with costochondral cartilage. Large defects left in a skull, even that of a young child, will not always close, and may later be a source of serious consequences. The defect is always exposed to trauma. It may, however, be protected with a metal cap, as described by Seifert.[65] All large bony fragments, except those definitely contaminated, should be replaced, even if completely loosened from all tissue at the time of operation.

In children, small depressions, areas of bone only slightly below the surface of the skull and depressions involving the nasal sinuses or the sinus sagittalis superior if they produce no symptoms, are often best treated without operation. Also, depressions only of the external table of the skull should be treated conservatively.

As a routine, the patients on whom this study is based were kept in bed for 4 weeks. The shortest stay in the hospital was less than 1 day, the longest 100 days, and the average 19 days. In many cases the patient was sent home after having spent less than 4 weeks in the hospital, and instructions were given to keep him in bed at home for a total of 4 weeks. Ice packs were placed on the head on entrance to the hospital. Patients suffering from pain and shock were treated with sedatives. Ether was given to 1 patient for convulsions, and stimulations were given to 1 patient. Tetanus antitoxin was given in compound fractures, deep lacerations or dirty wounds, to 10 patients (12.5 per cent). No medication was administered to 18 (22.5 per cent).

The patients were followed in the dispensary as long as possible. The shortest length of time of observation was less than 1 day; the longest, 6 years and 11 days; the average, 396 days. Sixty-nine patients had completely recovered at the last examination. Nine patients

63. Cushing, Harvey: Fractures of the Skull, in Williams, William: Keen Surgery, Philadelphia, W. B. Saunders Company, 1919, vol. 3, p. 86.

64. Hanson, A. M.; Hessly, Hanson, and Traeger: Reconstruction of Internal Table in Cranioplasty, Mil. Surgeon **50**:31 (Jan.) 1922.

65. Seifert, E.: Verlangt die Knochenlücke im Schadeldach einen abesondern Schutz, Zentralbl. f. Chir. **55**:910 (April 14) 1928.

(11.2 per cent) had serious sequelae when last examined, which were as follows: epilepsy, 1; paralysis of the left arm and enlargement of the left pupil, 1; weakness of the left leg, 1; the left leg weaker and smaller than the right, 1; a staggering gait, 1; paralysis of the right side of the face and troublesome pulsation in the right ear, 1, and headache, 3.

Healy [66] stated that in 3.5 per cent of children who are delinquent the condition is due to a trauma to the head. Strecker and Ebaugh [67] reported 30 cases of injury to the head in children with neuropsychiatric sequelae. Six of these children were improving, but the outlook for the 24 others was discouraging. Reichman [68] stated that not more than 0.5 per cent of fractures of the skull are followed by epilepsy, and that injury to the left side is more inclined to be followed by epilepsy than injury to the right side. Beekman [9] stated that less than 5 per cent of patients with fracture of the skull show permanent change; Pollak [62] thought that many unrecognized cases of fracture of the skull cause disorders of the brain later in life. Johnson [15] reported that 35.3 per cent of his 85 patients had serious sequelae. Stewart [8] reported that 55 per cent of 255 patients who recovered had unpleasant sequelae. English [69] reported that in more than 10 per cent of persons with injury to the head there is some mental impairment. Troell and Holmström [70] reported that 6 per cent of 152 patients were placed on the pay list for invalids in Sweden. Taft and Strecker [71] stated that less than 1 per cent of psychoses probably are due to trauma to the head. Orr [72] reported a case of jacksonian epilepsy that occurred 12 years after fracture of the skull. Smith and Emera [73] reported a case in which death occurred from fracture of the skull 11 years after the injury. Eager [74] reported a case of psychosis in a patient in the third decade of

66. Healy, William: A Review of Some Studies of Delinquents and Delinquency, Arch. Neurol. & Psychiat. **14**:25 (July) 1925.

67. Strecker, E. A., and Ebaugh, F. G.: Neuropsychiatric Sequelae of Cerebral Trauma in Children, Arch. Neurol. & Psychiat. **12**:443 (Oct.) 1924.

68. Reichman, V.: Ueber Entstehund und Häufigkeit epileptischer Krampfe nach Schadelbruchen, Deutsche Ztschr. f. Nervenh. **96**:260, 1927.

69. English, T. C.: The After Effects of Head Injuries, Lancet **1**:485, 1904.

70. Troell, Abraham, and Holmström, Per: Om skallfracturernas diagnosis, Svenska läk.-tidning. **24**:409, 1927.

71. Taft, A. E., and Strecker, E. A.: Psychosis Associated with Trauma of the Head, Arch. Neurol. & Psychiat. **14**:658 (Nov.) 1926.

72. Orr, T. G.: Late Results of Skull Fracture, J. Kansas M. Soc. **20**:162, 1920.

73. Smith, Sidney; and Emera, Mohammed: A Case of Fracture of the Skull Causing Death After Eleven Years, Brit. M. J. **3**:647, 1923.

74. Eager, Richard: Head Injuries in Relation to the Psychosis and Psychoneurosis, J. Ment. Sc. **66**:111 (April) 1920.

life, which perhaps was due to a fall in infancy. Cushing [75] reported a case of meningeal endothelioma in a patient in whom the symptoms developed 12 years after an injury to the head.

SUMMARY

1. Eighty cases of proved fracture of the skull in children, with 2 deaths (2.5 per cent), are discussed.

2. In all the cases, except 7, the treatment was conservative, without decompression.

3. Seven patients with depressed fractures and 3 patients with elevated fractures were treated without operation. All recovered.

4. Lumbar puncture should not be done for diagnosis when diagnosis can readily be made otherwise.

5. Children suffering from fracture of the skull should, in most cases, be treated conservatively.

5412 North Clark Street.

75. Cushing, H. W.: Cranial Hyperostosis Produced by Meningeal Endotheliomas, Arch. Neurol. & Psychiat. **8**:139 (Aug.) 1922.

TORSION OF THE OMENTUM

ITS CLINICAL IMPORTANCE *

JOHN H. MORRIS, M.D.
Assistant Visiting Surgeon, Fourth Division, Bellevue Hospital
Assistant Professor of Surgery, New York Postgraduate Hospital
NEW YORK

The great or gastrocolic omentum has never played a particularly prominent part in clinical surgery, its rôle generally being accepted rather as that of an inert structure with a function more or less vague and a relationship to clinical symptoms too remote to merit serious consideration in the diagnosis of abdominal conditions. Of late, however, in consequence of the rapid accumulation of reports and data relative to torsion of the omentum, this structure has assumed some degree of clinical importance, and it is significant of this altered status that at least one modern surgical text [1] has given prominent consideration to this disease in the differential diagnosis of acute appendicitis. Accordingly, the conclusion now seems justified that, under certain conditions, the omentum possesses inherent potentialities capable of initiating abdominal crises which demand differentiation from other and more familiar acute abdominal lesions.

In an abstract physical sense, torsion of a structure may be defined as a turning or twisting about its long axis. Two varieties may be recognized: (1) the unipolar, in which the structure is fixed at one extremity while its distal end swings free in response to any forces acting on it; as an example of this type may be mentioned the plumb line, the weighted end of which is suspended in midair; (2) the bipolar, in which the structure is fixed at two points with the intervening portion left free to twist axially; a handkerchief suspended by two diagonally opposed corners, permitting rotation between, has aptly been suggested by Picquet [2] as an example of this variety.

Clinically, the most familiar examples of torsion are encountered with ovarian cysts, the testicle and the spleen, in which a comparatively heavy, solid mass attached to a long thin pedicle provides ideal mechanical conditions for an axial twist of the unipolar type. A rare instance of torsion involving the lesser omentum and producing symptoms sug-

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* Read in abstract before the New York Celtic Medical Society, Jan. 16, 1930.

1. Lewis: Practice of Surgery, Hagerstown, Md., W. F. Prior Company, Inc., 1929, vol. 7, p. 20.

2. Picquet: Bull. et mém. Soc. anat. de Paris **85**:717, 1910.

gestive of perforation of the stomach was recorded by Hartwell,[3] while Studebaker [4] mentioned fifty-three cases of torsion of the appendices epiploicae which gave rise to acute abdominal manifestations.

Owing to its character and environment, torsion arising in the great omentum is somewhat more complex, since both the unipolar and bipolar types of rotation may occur. With the omentum swinging normally free from its attachment to the stomach and colon, its dependent portion is acted on by a variety of forces within and without the peritoneal cavity. Hence any factor, such as cysts, tumors, inflammatory tumefaction or circumscribed fibrosis, which produces a local increase in the density of this dependent portion may contribute toward unipolar torsion. More frequently, however, the well known tendency on the part of the omentum to attach some portion of its free border to another intra-abdominal structure accounts for the second fixed point essential to bipolar torsion and in consequence, this type of torsion preponderates when this organ is concerned.

Marchette was credited by Aimes [5] with reporting the first case of torsion of the omentum, in 1851, but Oberst,[6] in 1882, presented the first available record of this condition, which was supplemented by that of Demons [7] in 1893. These cases were all associated with right inguinal hernia, a fact which seemed to establish this complication as a predisposing factor until Eitel,[8] in 1899, reported the first case of pure abdominal torsion occurring independent of the presence of hernia.

In 1905, Corner and Pinches [9] compiled from the literature the reports of fifty-one authentic cases, to which they added three of their own, making a total of fifty-four. From the information thus assembled they contributed the most complete study of this subject made before or subsequent to that year. Analysis of the data obtained from this series indicated that torsion of the omentum is a disease of middle life, 78 per cent of the cases having occurred in persons between the ages of 35 and 55, although the youngest patient was 17 (a case reported by Broca [10]) and the oldest, 79 (a case reported by Weiner [11]). Males were more commonly affected than females, in the proportion of 69.7 to 32 per cent. The importance of inguinal hernia as a predisposing factor was shown by the fact that it was present in 90 per cent of the

3. Hartwell: Ann. Surg. **61**:626, 1915.
4. Studebaker: J. Iowa M. Soc. **11**:350 (Sept.) 1921.
5. Aimes: Progrès méd. **34**:425, 1919.
6. Oberst: Zentralbl. f. Chir. **27**:441, 1882.
7. Demons: Rev. de chir. **13**:159, 1893.
8. Eitel: M. Rec. **55**:715 (May 20) 1899.
9. Corner and Pinches: Am. J. M. Sc. **130**:314, 1905.
10. Broca: Bull. et mém. Soc. de chir. de Paris **26**:170, 1900.
11. Weiner: Ann. Surg. **32**:648, 1900.

cases. Furthermore, these herniae presented certain common features which were regarded as of definite etiologic significance; namely, they were usually in the right inguinal region, scrotal in type, of long duration and easily reducible, and they almost invariably contained omentum.

Six, or 10 per cent, of the cases in this series were of the so-called pure abdominal type; i. e., the torsion developed within the abdomen and was not associated with any type of hernia. The latter group attracted particular interest, not only because of the absence of an associated pathologic process and the obscurity of the mechanism of their production, but also because of the diagnostic difficulties that they created. Interest in this phase of the subject has been sustained to the present time. In 1928, McWhorter,[12] in a study of this type of torsion, collected twenty-four of these cases, to which he added two of his own.

In the early cases, classification was rendered superfluous by reason of the fact that all of them could be included in a single category; namely, the hernial. Subsequent reports, however, brought out, in addition to Eitel's case, instances of pure abdominal, nonhernial torsion together with certain subvarieties of the hernial type itself, thus calling for some form of grouping.

The effort to provide such a grouping competent to cover all the observed types of torsion has resulted in a variety of classifications which are, in many instances, as elaborate and complex as they are numerous. In spite of the fact, however, that they appear to differ in nomenclature and arrangement, all these classifications are essentially identical in their fundamental recognition that all cases of torsion fall into one of two main groups: (*a*) torsion with an associated pathologic process, e. g., hernia, and (*b*) torsion without demonstrable associated disease. No consideration has therefore been given to the conception that torsion may be complete or incomplete, and no attempt has been made to distinguish between the acute and chronic varieties of this condition. It is therefore my purpose to emphasize the clinical import of the latter phases of torsion of the omentum, and, as a basis for my description it is advisable to adopt the following modified classification, which includes them in their conceived relationship to the other forms.

A. Complete torsion
- 1. Acute torsion with hernia
  - (*a*) Hernial
  - (*b*) Hernial and abdominal (combined)
- 2. Acute torsion without hernia (purely abdominal)
  - (*a*) Primary or idiopathic (unipolar)
  - (*b*) Secondary (unipolar and bipolar)

12. McWhorter, G. L.: Torsion of Omentum Without Hernia, Arch. Surg. **16**:569 (Feb.) 1928.

B. Incomplete torsion
1. Chronic recurring torsion (unipolar and bipolar)
   (*a*) With hernia
      1. Hernial
      2. Combined
   (*b*) Without hernia
      1. Primary
      2. Secondary

Richardson,[13] in discussing torsion of the omentum, unconsciously but accurately defined the scope of the terms "complete" and "incomplete" as applied to this classification. "True torsion should include," he stated, "only those cases in which twisting of the omentum on itself has caused sufficient obstruction of the circulation to produce evidences of strangulation." According to my conception, circulatory obstruction of this degree presupposes organic changes in the tissues, progressing from simple congestion and ĕdema to thrombosis, infarction and gangrene. It may be assumed also that such changes are permanent, and that they lead to symptomatic manifestations which are acute and from which there is no spontaneous retrogression. This form of torsion therefore fulfils, within the meaning of the classification, the conception of the "complete" type, and under it obviously must also be included those lesions which are acute by reason of the pathologic process produced thereby.

But to continue with Richardson's statement: "The matting of the omentum into a mass or ball which, although a part of the pathological process producing torsion, should not, by itself, be included under this term."

According to the classification given the pathologic process thus described accurately pictures the condition for which the term "incomplete torsion" was adopted. In my opinion, this process which is "a part of the pathological process producing torsion" differs only in degree from so-called "true torsion," and its exclusion from this category must be based solely on the assumption that it plays no part, immediate or remote, in the production of symptoms.

On the contrary, it is possible to demonstrate by clinical evidence, to be presented herein, that the omental changes grouped under the term "incomplete torsion" are responsible for distinctive clinical manifestations which justify the inclusion of this group in any comprehensive classification of torsion of the omentum.

The analysis by Corner and Pinches,[9] of fifty-three cases of torsion of the great omentum occurring prior to 1905, emphasized the possibilities of this condition and served to familiarize its clinical and

13. Richardson, W. W.: Torsion of the Great Omentum, J. A. M. A. **48**:1590 (May 11) 1907.

pathologic characteristics. The ensuing period has been marked by an added keenness in the recognition of the disease and in consequence thereof the interval from 1905 to 1929 has witnessed a rapid accumulation of authentic, accurately observed cases. In view of the fact that the latter cases present significant new data relative to symptomatology and diagnosis, and as no systematic study of these data has been carried out during this period, it appears advisable to supplement the work of Corner and Pinches by an analytic study of the cases reported in the literature from 1905 to 1929 inclusive.

A careful search of the literature of this period revealed a total of 164 cases, 161 of which were sufficiently complete to permit accurate analysis and classification. Including Corner and Pinches' 1905 series, 217 cases of torsion were therefore available for study.

In my 1929 series, 101 (62.7 per cent) were males and 60 (37.2 per cent) were females; in 3 instances the sex was not indicated. The

TABLE 1.—*Age Incidence by Decades of Torsion of the Great Omentum*

Age, Years	Cases, Number	Cases, per Cent
0 to 10	3	1.8
10 to 20	5	3.1
20 to 30	29	18.3
30 to 40	43	27.2
40 to 50	40	25.3
50 to 60	29	18.3
60 to 70	8	5.06
70 to 80	1	0.63

average age in this series was 45.3 years. The oldest patient (a case reported by Brown [4]) was 80 and the youngest (a case reported by Lawrence [15]), 3 years of age. Speese [16] recorded a case of torsion in a patient aged 5 years, Coen [17] in one aged 5, Erdmann [18] in one aged 14 and Strauss [19] in one aged 15. Table 1, in which age is tabulated by decades, indicates that the highest incidence of the disease (43, or 27.2 per cent of cases) occurred during the fourth decade.

For purposes of analytic study, the cases of this series have been grouped in accordance with the classification given and it is therefore essential to clarify and define accurately the scope of the terms employed therein.

The term "torsion" itself, as applied to the great omentum, implies the rotation of that structure about its long axis with the resulting

14. Brown: Brit. M. J. **1**:183 (Jan. 30) 1926.
15. Lawrence: South. M. J. **18**:423 (June) 1925.
16. Speese: Internat. Clin. **30**:246, 1920.
17. Coen: Policlinico (sez. chir.) **35**:252 (May) 1928.
19. Strauss: Beitr. z. klin. Chir. **148**:36 (Nov. 23) 1929.
18. Erdmann: Ann. Surg. **88**:423 (June) 1925.

formation of a narrow neck or pedicle somewhere in its continuity. Disregarding for the moment the causative factors involved in this process, it is obvious that subsequent developments in the involved omentum will be governed by the degree and permanence of the circulatory impairment at the site of the twist. Knowing the character, mobility and environment of the great omentum, it is not difficult to accept the fact that temporary twists of this sort occur and recur frequently; but under ordinary circumstances, spontaneous readjustment takes place before extensive damage is accomplished. Nevertheless, these temporary twists are evidenced clinically through the sympathetic nervous system by certain vague, transitory abdominal symptoms, and their tendency to recur is responsible gradually, through vascular changes, for slight local omental thickening of fibrosis—the so-called "omental ball."

On the other hand, the torsion occasionally assumes a more definite intensity and permanence, spontaneous readjustment fails to occur, and the lesion becomes progressive. The early congestion at the site of the twist is soon succeeded by edema which intensifies the existing vascular obstruction, and the sequence of thrombosis, infarction and gangrene supervenes in the distal segment of twisted omentum.

Thus the degree of vascular obstruction becomes the basis on which one distinguishes primarily two main varieties of torsion, the complete and the incomplete. The complete type is dynamic and acute, with a rapidly progressive pathologic process and symptoms; the incomplete form is static, with chronically recurrent symptoms of an ill defined nature accompanied by rather indefinite pathologic changes of a non-progressive type.

The presence or absence of hernia associated with the torsion becomes the basis on which the cases of complete torsion are further divided under two main subgroups; namely (1) acute torsion with hernia and (2) acute torsion without hernia. The former includes all cases in which (*a*) the torsion is confined to the hernial sac (the hernial type [fig. 1*a* and *b*]); (*b*) the torsion within the hernia "runs up" the omentum into the abdomen, producing secondary intra-abdominal pedicles and contorted masses (the combined type [fig. 1*c* and *d*]), and (*c*) the contorted mass is completely intra-abdominal, but the presence of a hernial sac, even though empty, is regarded as sufficient evidence of its etiologic relationship. (fig. 1*e*).

The latter type, acute torsion without hernia, occurs independently of hernia and is purely abdominal, topographically and etiologically. Its subtypes comprise: (1) a unipolar form in which no direct mechanical factor predisposing to torsion can be demonstrated; it therefore is classed as the "primary" or idiopathic" type (fig. 1*f*); (2) unipolar and bipolar varieties in which definitely associated mechanical factors are

responsible for the initial twist—the secondary type (fig. 1*g* and *h*). These factors may be (*a*) a tumor, a cyst or other causes of increased density in the dependent omentum favoring the unipolar type of torsion or (*b*) the adhesion of the omental tip to the parietes or an adjacent intra-abdominal organ, thus providing a second fixed point which makes possible the bipolar type of torsion.

Under the classification of incomplete torsion are grouped the chronic recurring varieties, but owing to the fact that experience with this type

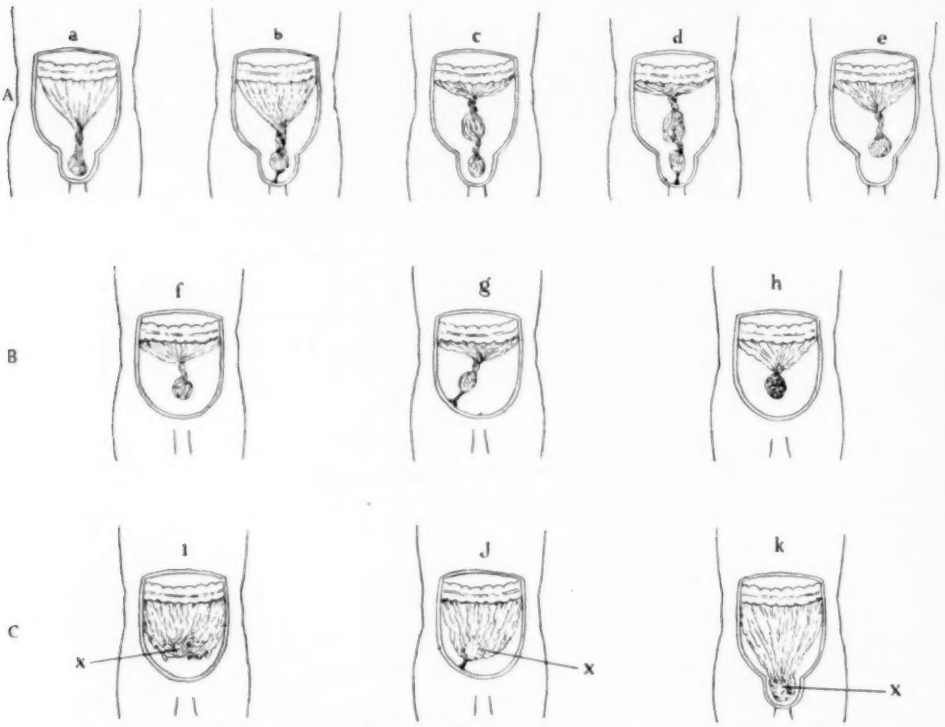

Fig. 1.—*A*, various types of torsion of the omentum: *a*, acute hernial, unipolar; *b*, acute hernial, bipolar; *c*, acute combined, unipolar; *d*, acute combined, bipolar; *e*, acute combined (sac empty); *B*, types of complete torsion without hernia: *f*, acute primary, unipolar; *g*, acute secondary, bipolar; *h*, acute secondary, due to tumor or cyst; *C*, types of incomplete torsion with and without hernia: *i*, primary abdominal (*x*, omental ball); *j*, secondary abdominal, and *k*, hernial.

of case has been comparatively limited, the subvarieties are not so well defined as those of complete torsion. There are discernible, however, certain similar anatomic and mechanical factors common to both the complete and the incomplete types of torsion, thus suggesting that analogous subtypes should occur under each of them. In order, therefore, to carry the present classification to completion and so anticipate

the results of further observation, this analogy has been drawn on for the purpose of achieving a satisfactory terminology for the subtypes under incomplete torsion.

ANALYSIS OF THE CASES

Of the total of 164 cases in the 1929 series, 161 showed the complete type of torsion. Of these, 81 (50.3 per cent) were associated with hernia, while 77 (47.8 per cent) were of the purely abdominal or nonhernial type. When these figures are compared with those of Corner and Pinches' 1905 series in which 11 per cent (compared with 47.8 per cent) were purely abdominal and 89 per cent (compared with 50.3 per cent) were associated with hernia, it is evident that the period from 1905 to 1929 has been marked by a striking increase, relative as well as absolute, in the purely abdominal type of case. This increase, however, is obviously more apparent than real and is to be interpreted as the result of a combination of factors: 1. The impression gained from the early cases, that hernia was an essential etiologic accompaniment of torsion, was dispelled, in the light of wider experience, to permit the inclusion of many cases of pure abdominal torsion previously overlooked. 2. The increasing popularity and efficiency of the radical surgical treatment for inguinal hernia was unquestionably responsible for the elimination of potential sources of hernial torsion in many instances. 3. The substitution of surgical methods of treatment for the older method of taxis, which of itself accounted for numerous cases of torsion with hernia, further reduced the incidence of this type.

ACUTE TORSION WITH HERNIA

Under the heading of acute torsion with hernia are placed eighty-one (50.3 per cent) of the cases of the series, and within this group two subvarieties are to be distinguished: (1) the intrasacular, comprising 16 (19 per cent) of the group, in which the torsion was limited to the hernial sac (fig. 1), and (2) the combined, numbering sixty-five (80 per cent) of the group in which (*a*) the torsion was not limited to the hernial sac but extended upward to produce secondary masses and pedicles in the abdominal portion of the omentum (fig. 1), or (*b*) the contorted omental mass was found entirely within the abdomen but an accompanying hernial sac, although empty, was accepted as evidence of the hernial origin of the torsion. Nevertheless, Smythe,[20] Hedley,[21] and Mesa,[22] in reporting cases of this type, emphasized that "the torsion had no connection with the hernia," thus evidently implying a primary

20. Smythe: Surg., Gynec. & Obst. **3**:531, 1906.
21. Hedley: Brit. M. J. **2**:1246, 1911.
22. Mesa: Semana méd. **30**:949, 1923.

abdominal origin of the torsion and eliminating the hernia as an etiologic factor. It is, however, clear from clinical experience with the hernial types of torsion that spontaneous reduction of the hernial mass of omentum occurs with considerable frequency, and that the torsion is thereupon found to be entirely intra-abdominal, the sac is empty, and no evident anatomic connection exists between the two parts. The existence of a hernia in conjunction with an intra-abdominal torsion is therefore accepted as establishing the hernial nature of the latter, and the demonstration of an actual anatomic connection is not essential to its classification under the combined form of torsion.

Among the herniae associated with acute hernial torsion are found representatives of all the external abdominal varieties. Their types and their distribution in the series are indicated in table 2.

Therefore, of eighty-one cases of acute hernial torsion, the accompanying hernia was inguinal in 93.7 per cent, umbilical in 24 per cent,

TABLE 2.—*Types and Distribution of the Accompanying Herniae in Acute Hernial Torsion*

Type of Torsion	Inguinal Hernia, Right	Inguinal Hernia, Left	Umbilical Hernia	Ventral Hernia	Femoral Hernia, Right
Intrasacular	8	5	2	1	0
Combined	58	5	0	1	1
Total	66	10	2	2	1
Percentage	(81.4)	(12.3)	(2.4)	(2.4)	(1.2)

ventral in 2.4 per cent and femoral in 1.2 per cent. Of those herniae which occurred bilaterally; namely, inguinal and femoral, the right side was implicated in 87 per cent and the left side in 13 per cent.

The duration of the hernia is of some interest from an etiologic standpoint. It was referred to in forty-seven of the histories in this group. In thirty-one of these, the average duration was seventeen years, the shortest being two and the longest being forty years; while in the remaining sixteen cases this period was described as "long standing," "since childhood," "many years." etc. In seven of the cases, torsion developed in association with herniae which had recurred after operation. In the cases of Bufalino [23] and Cavara [24] the herniae had recurred after the second operation, the torsion being related to the second hernial recurrence. In Reidel's [25] case, torsion occurred in conjunction with a hernia which recurred after treatment by means of a truss. Grant [26] cited an instance in which torsion was observed at the site of a hernia apparently cured by the use of a truss two years previously.

23. Bufalino: Policlinico (sez. chir.) **31**:289 (June) 1924.
24. Cavara: Semana méd. **1**:223 (Jan. 24) 1929.
25. Reidel: München. med. Wchnschr. **52**:2257 (Nov. 21) 1905.
26. Grant: Colorado M. J. **5**:195, 1908.

In addition to their long duration, these herniae, particularly the inguinal type, disclosed other common characteristics that may have a distinct bearing on the production of omental torsion. They were usually of the well developed scrotal type, easily reducible and, as a rule, giving rise to no pain up to the onset of the acute attack. The contents of the sac were almost invariably omentum, and reduction had been practiced habitually by the patient or the attending physician over a period of years.

The stature or make-up of the patient was referred to in twenty-four of the cases; in twenty-one of these, the patient was described as "obese," "fat" or "robust," while only three were classed as "thin." The preponderance of the obese type in this small group scarcely justifies the conclusion that obesity is a characteristic of the patient with torsion, but it is probable that more careful observations would have established that the majority of these persons are well nourished. This consideration is of some etiologic importance and will be referred to again under that heading.

The onset of the immediate attack was described as acute in forty cases and subacute in ten; in thirty-one of these the onset was attributed to a definite exciting cause. In ten instances [27] the attack began during or shortly after lifting a heavy object or followed extreme muscular strain. Heavy falls initiated the symptoms in four cases,[28] sharp blows on the abdomen were described as causative in seven cases,[29] and in three cases the attack followed the forcible reduction of a scrotal hernia.[30] In Verbely's [31] case the attack occurred while the patient was bowling, in Schönwerth's [32] case while the patient was stretching across a table and in Thevenard's [33] case during the course of a bicycle ride. Patel and

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27. McWhorter (footnote 12). Reidel (footnote 25). Kohler: Arch. f. klin. Chir. **3**:514, 1918. Mullen: Surg., Gynec. & Obst. **40**:635 (May) 1925. Siefert: quoted by Mast: Med. Welt. **2**:128, 1928. Nixon: Texas State J. Med. **20**:659 (April) 1925. Lapeyre: Bull. et mém. Soc. de chir. de Paris **33**:192, 1907. Lucene and Mondor: Bull. Soc. anat. de Paris **6**:346, 1911. Allen: J. Iowa M. Soc. **14**:549 (Dec.) 1924. Wildenskov: Ugesk. f. læger **82**:40, 1920.

28. Tuffier: Bull. et mém. Soc. de chir. de Paris **32**:301, 1906. Uffreduzi: Gior. d. r. Accad. di med. di Torino **18**:45 and 106, 1912. Syme: Intercolon. M. J. Australasia, 1902, p. 444. Hedley (footnote 21).

29. Studebaker (footnote 4). Eitel (footnote 8). McWhorter (footnote 12). Bazy, quoted by Aimes (footnote 5). Cernezzi: Clin. chir. **17**:654, 1909. Lucene and Mondor (footnote 27, eighth reference). Hadda: Arch. f. klin. Chir. **92**:843, 1910.

30. Arcoleo: Morgagni, 1909, p. 422. Hotchkiss: Ann. Surg. **45**:759, 1907. Albrecht: Gynäk. Rundschau **5**:259, 1911.

31. Verbely: Wien. med. Presse **48**:1561, 1907.

32. Schönwerth: Beitr. z. klin. Chir. **48**:118, 1906.

33. Thevenard: Paris chir. **3**:38, 1911.

Santy,[34] Morestin[35] and Taddei[36] reported cases in which drastic catharsis seemed to initiate the trouble. In Pretzsch's[37] case, symptoms developed while the patient was dancing.

Whether ushered in in this manner or, as frequently occurred, developing independent of a demonstrable exciting cause, the onset of the acute attack was usually first expressed as a change in the character of the hernia which, having previously been easily reducible and painless, was suddenly observed to be irreducible, tender, firm and sometimes increased in size. The pain that accompanied these changes was, in a few instances (intrasacular types), confined to the scrotum; as a rule, however, it was first manifested generally over the abdomen before eventually localizing to the right, of the umbilicus or in the right lower quadrant (combined types). This pain was usually of a severe, colicky, prostrating type, which "doubled up" its victims and at times gave the impression of an acutely perforated viscus. Nausea and vomiting followed in 45 per cent of the cases, elevation of temperature (from 99 to 102 F.) in 58 per cent and acceleration of pulse in 26 per cent. In twelve cases in which blood counts were recorded there was a leukocytosis ranging from 12,000 to 19,000 white blood cells. Tenderness was almost invariably elicited to the right of the umbilicus and in the right lower quadrant of the abdomen. Spasm of the right rectus muscle was a frequent observation, usually preventing accurate palpation of the structures beneath. In twenty of the cases, however, a large firm, tender mass could be felt in the right lower quadrant of the abdomen soon after the onset of the attack. In a large proportion of these cases the mass was freely movable. Mesa's[22] patient was able to secure relief from pain by displacing the tumor upward by means of external pressure. In two cases[38] the discovery of the mass per vaginam led to a diagnosis of twisted ovarian cyst. It isolated instances, inconstant symptoms, such as dysuria, diarrhea and jaundice, were observed during acute attacks; in one case, bleeding from the stomach was noted by Gillette,[39] while Wildenskov's[40] patient experienced intestinal hemorrhages.

The gross pathologic process encountered in conjunction with these clinical manifestations was essentially similar throughout the group of cases of torsion with hernia, although considerable variation in the degree and extent of the process was described.

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34. Patel and Santy: Lyon chir. **10**:35, 1913.
35. Morestin: Bull. Soc. anat. de Paris **89**:85, 1914.
36. Taddei: Riforma med. **37**:289 (March 26) 1921.
37. Pretzsch: Beitr. z. klin. Chir. **48**:118, 1906.
38. Tuffier (footnote 28, first reference). Cinaglia: Gazz. d. osp. **34**:859, 1913.
39. Gillette: Am. J. Obst. **63**:112, 1911.
40. Wildenskov (footnote 27, tenth reference).

Typical of the intrasacular variety was a long thin pedicle with a variable number of twists occupying the inguinal canal and often reaching as high as the internal ring. This pedicle communicated in the distal hernial sac with the thickened congested or gangrenous mass of omentum which was sometimes adherent at one or more points in the sac (bipolar), but was as frequently found free therein with no second fixed point present (unipolar).

In the more common combined variety, the twisting process "ran up" through the inguinal pedicle to produce secondary masses and pedicles in the abdominal portion of the omentum. In some extreme cases this progressive twisting process resulted in a series of two or three intra-abdominal masses with intervening pedicles, and sometimes involved the entire omentum up to its origin at the transverse colon. More characteristic of this combined type, however, was the single intra-abdominal omental mass usually situated in the right lower quadrant or the right umbilical region. Proximally, it presented a twisted pedicle extending up the omentum for a variable distance, while distally it communicated with the scrotal mass by way of the inguinal pedicle.

An important and significant characteristic of combined torsion is the freedom of the inguinal pedicle in its course through the inguinal canal and the internal ring. This feature emphasizes the fact that the circulatory changes distal to this pedicle are due to its intrinsic twisting mechanism rather than to the constricting effect of the contiguous parts of the canal. On the other hand, strangulated omental herniae, which at operation may closely resemble scrotal torsion in most other respects, present definite evidence of constriction at the internal ring or within the canal to account for the distal vascular changes.

Differentiation between these two conditions is of more than academic interest. Demons [7] operated on an apparently uncomplicated strangulated omental hernia and removed a gangrenous omental mass the pedicle of which was amputated at the internal ring. The persistence of abdominal symptoms led subsequently to laparotomy, which disclosed a peritonitis secondary to the gangrenous intra-abdominal portion of a previously unrecognized combined torsion of the omentum. Trinkler [41] operated in a similar case, but recognizing the significance of a free, nonconstricted pedicle, he extended his incision and removed an intra-abdominal mass of twisted omentum.

It is therefore evident that the finding of a supposed omental hernia, the scrotal mass of which presents evidence of vascular obstruction combined with a free, nonconstricted inguinal pedicle, should arouse

41. Trinkler: Deutsche Ztschr. f. Chir. **75**:269, 1904.

suspicion of combined torsion with the possibility of an associated intra-abdominal pathologic process.

The gross structural changes encountered in the omental masses thus produced varied with the degree of circulatory obstruction produced at the site of the twist, but were not necessarily proportional to the number of twists present. In many instances, these changes had not proceeded beyond the stage of thickening, fibrosis and "omental ball" formation, the normal color and lobulation of the omentum being preserved. In others, in which circulatory obstruction was more definite, edema, congestion thrombosis, infarction and even gangrene had developed, the normal lobular structure was lost and the mass assumed a beefy consistency with a color varying from red to violet or black.

These omental changes were usually responsible for, and accompanied by, evidences of reaction on the part of the peritoneum to the presence of the intra-abdominal mass. The nature of this reaction was determined by the severity of the omental process. In many instances numerous fine adhesions between the mass and such contiguous structures as the abdominal wall, intestine or uterus betokened a low grade inflammatory irritation. The filmy character of these adhesions indicated a recent formation and distinguished them definitely from the more dense, permanent omental adhesion which was often coexistent as the "second fixed point" in the bipolar type of torsion. In the more acutely involved cases free fluid was a common observation; in twenty-five (30 per cent) of the cases, considerable quantities of this fluid, serosanguineous to frankly bloody, was described as a prominent feature at laparotomy.

From the data thus accumulated, a composite clinical picture of a hypothetic case of torsion with hernia may be constructed. The patient is usually an obese, robust or well nourished man in middle life who for years has had hernia, most probably of the right inguinoscrotal type. During this period the hernia has not caused pain and has been easily reducible. Suddenly, and often following a severe physical strain or muscular effort, it becomes irreducible, painful and increased in size. The pain becomes severe, colicky or prostrating, and gradually extends up over the abdomen to localize to the right of the umbilicus or in the right lower quadrant. During the next twenty-four to forty-eight hours, the patient's condition becomes progressively worse, with a rising temperature, nausea, vomiting and an accelerated pulse rate. Abdominal examination discloses tenderness and rigidity on the right side. A large, tender, movable mass suddenly makes its appearance in the right lower quadrant. Blood counts show a moderate leukocytosis.

The preoperative diagnosis usually rests between acute appendicitis with abscess and a strangulated inguinal hernia. If the latter is made,

herniotomy exposes in the hernial sac a congested or gangrenous mass of omentum, the omental pedicle of which extends upward to the internal ring. This pedicle may present evidence of twisting, but of even greater significance is the absence of any point of constriction within the canal or at its internal ring to account for the circulatory changes below. It is this combination of a free pedicle with distal circulatory disturbances which suggests the possibility of intra-abdominal extension of the torsion (combined type). Recognition of the importance of these relations will lead, in a certain percentage of cases, to herniolaparotomy with indications characteristic of the combined type of torsion.

ACUTE TORSION WITHOUT HERNIA

In seventy-seven (47.8 per cent) of the cases in the series, torsion occurred independent of the presence of hernia and was therefore purely abdominal in location. Within this group the cases were divided into two subtypes: (1) the primary or idiopathic and (2) the secondary. Forty-one (53.2 per cent) of the cases were included under the heading of primary torsion because they appeared to be of spontaneous or idiopathic origin; i.e., there was no evidence of an antecedent change in the omentum or in its environment to account for the initiation of the twisting process. On the other hand, thirty-six (46.7 per cent) of the cases of this group presented definite evidence of preexisting pathologic factors which were mechanically competent to precipitate a torsion and they were accordingly classed as the secondary type. These factors were of three distinct types: (*a*) inflammatory processes in structures adjacent to the omentum; in thirteen of the thirty-six cases of secondary torsion, the appendix showed evidence of acute, subsiding or comparatively recent inflammation (the significance of this fact will be further touched on under etiology); (*b*) tumor, cyst or other areas of increased density in the free, dependent portion of the omentum. The relation of this condition to torsion was discussed under mechanics of unipolar torsion, and three cases illustrative of this type were included in this series; in Tuffier's case, a hydatid cyst of the omentum was apparently responsible for the inception of the twist; Speese reported a case in a child aged 5 years, and Lawrence a case in a child aged 3, in both of whom an omental cyst of congenital origin appeared to be the inciting stimulus to an acute torsion; (*c*) adhesion of a portion of the free margin of the omentum to some adjacent structure. As pointed out under bipolar torsion, there is thus established the second fixed point which forms, with the normal fixed point of the amentum at its origin, an axis about which the adjacent portions of the omentum tend to rotate. In nineteen of the thirty-six cases of the secondary type, torsion had occurred between two such fixed points and was therefore of the bipolar

variety. Various intra-abdominal structures shared with the omentum in the formation of this "second fixed point." In five cases the distal omental adhesion was at the stomach, ascending colon or cecum; in four cases the point of fixation was somewhere on the anterior abdominal wall; in three cases an omental tongue had attached itself to an ovarian cyst which had itself undergone torsion, the omental torsion evidently being a secondary process.[42] In the pelvis, the uterus—pregnant in one instance—served as the "second fixed point" in three cases while in three other cases an inflamed right tube was responsible. Luckett [43] operated on a patient in whom a tongue of omentum, tied off with the sac during a previous operation for inguinal hernia, had become the "second fixed point" of a bipolar torsion.

The gross omental changes encountered in the intra-abdominal types of torsion were, in principle, similar to those described under the heading of torsion with hernia. In general, however, the damage to the tissues was more extensive in the former, in which red infarction was frequent and actual gangrene not uncommon. The unipolar types presented the largest masses, as a rule, and in a considerable proportion the entire omentum was involved up to a short thin pedicle at the transverse colon (case 1). In the bipolar varieties the pedicles tended toward greater length and were occasionally double, and the masses were much smaller (case 2). Perietoneal irritation was evidenced, as in the hernial cases, by fresh filmy adhesions between the mass and surrounding structures, while large quantities of free fluid were usually found when the peritoneal cavity was opened.

It is of interest to note that in all of the aforementioned types of secondary torsion the omental mass was almost invariably located to the right of the midline and usually in the right lower abdominal quadrant. The same tendency toward localization on the right side was observed in the hernial cases and was accounted for by the predominance of right inguinal hernia over all other types. In the nonhernial group, however, it is evident that hernia no longer plays a part and that another explanation for this rather constant right-sided localization must be sought. Two factors suggest themselves: 1. It is a matter of common clinical experience that the right margin of the omentum is the more mobile, that it usually hangs well down on the right side and that the left margin is commonly found curled up in the left hypochondrium. It is therefore obvious that the right side presents conditions which favor

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42. Potherat: Bull. et mém. Soc. de chir. de Paris **34**:922, 1908. Frederick: Am. J. Obst **56**:742, 1907. Payr: Arch. f. klin. Chir. **68**:501, 1902.

43. Luckett, W. H.: Torsion of the Greater or Gastrocolic Omentum, J. A. M. A. **54**:1364 (April 23) 1910.

torsion of the unipolar variety. 2. The right side, and particularly the right lower quadrant, of the abdomen is a fertile field for the development of acute and chronic inflammatory processes: cholecystitis, gastric and duodenal ulcer and especially appendicitis. These conditions lead frequently to inflammatory fixation of a portion of the free margin of the adjacent omentum (second fixed point) in this region and thus predispose toward the bipolar type of torsion.

The clinical manifestations of the nonhernial type of torsion paralleled closely those of the hernial type, with the single exception that the former, in the absence of associated hernia, presented symptoms confined to the abdomen. Among the predisposing clinical evidences, the stature or make-up of the patient appeared to have a more definite bearing than in the hernial group. In thirty-four (44 per cent) of the cases of the former group, this feature was referred to, and in thirty-one the patient was classed as "obese," "stout" or "well nourished." Only three patients were described as "thin." In twenty-seven cases (35 per cent) the past histories disclosed evidence of vague intermittent digestive disturbances extending over a period of months or years which, in the light of final developments, could be interpreted as of omental origin. As already shown, in thirty-one cases the onset of the acute attack was related to a definite exciting cause: Ten cases followed heavy lifting or severe muscular effort, seven occurred after blows on the abdomen, while in four cases the onset was variously attributed to horseback riding,[44] violent coughing,[45] stooping over a tub[46] and being violently whirled about while wrestling.[47] The immediate attack was acute and severe in onset in 55 cases (71 per cent) and subacute in twenty-two (28 per cent). Characteristic symptoms accompanying the attack were nausea and vomiting, (42 per cent) elevation of temperature (70 per cent), leukocytosis (44 per cent) and acceleration of the pulse rate (41 per cent). Muscular rigidity and tenderness were constantly found to the right of the umbilicus or in the right lower quadrant. In thirty cases (38 per cent) a definite intra-abdominal mass was palpated soon after the onset of the attack, and the fact that a diagnosis of acute appendicitis or abscess of the appendix was made preoperatively in forty-nine cases (63 per cent) is indicative of the character and location of this mass.

The clinical and pathologic characteristics of acute torsion without hernia are well exemplified by the following two cases. Both belong to the secondary group of torsion. Case 1 represents a unipolar torsion

44. Ill: Am. J. Obst. **56**:742, 1907.
45. Adler: Arch. f. klin. Chir. **83**:250, 1907.
46. Ronald: Brit. M. J. **1**:811 (May 4) 1929.
47. Cullen: Bull. Johns Hopkins Hosp. **16**:401, 1905.

associated with an inflammatory process outside the omentum and case 2 illustrates the effects of the "second fixed point" in its relation to bipolar torsion.

REPORT OF CASES

CASE 1.—C. K.,[48] a white man, aged 28, a clerk, was admitted to the Postgraduate Hospital on Jan. 28, 1926, in an acute condition. Two days previously, he had suddenly been seized with severe generalized abdominal pain accompanied at the onset by nausea and vomiting. Within the following twelve hours the pain, undiminished in severity, localized in the right lower quadrant, and there remained continuous up to the time of admission to the hospital; it did not radiate. There was no dysuria, polyuria or diarrhea, and no history of trauma or muscular strain was obtained.

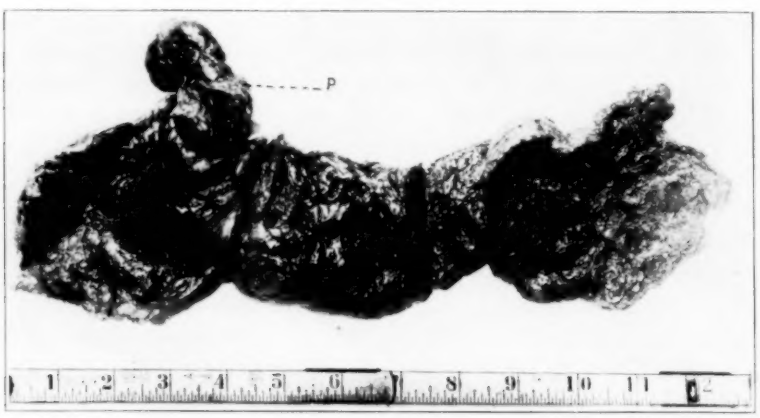

Fig. 2.—Specimen removed from patient in case 1, showing pedicle (*P*), partially untwisted, and gangrenous mass of omentum.

The previous history was unimportant. He had never experienced an attack similar to the present one, had never noted any form of digestive disturbance, and had never suffered from any type of acute or chronic illness.

The patient was short, thick-set and obese, weighing over 200 pounds (90 Kg.). On his admission to the hospital, his rectal temperature was 102 F. and his pulse rate 110. The abdomen was tympanitic throughout, while generalized tenderness and rigidity precluded the possibility of detecting an intra-abdominal mass. Both rigidity and tenderness seemed more marked over McBurney's point. The presence of free fluid could not be demonstrated by percussion and palpation. Urinalysis yielded negative results. A blood count showed 16,600 white blood cells with 83 per cent polymorphonuclears and 17 per cent lymphocytes. The preoperative diagnosis was ruptured appendix with general peritonitis.

Operation.—A right rectus muscle splitting incision was made. When the peritoneum was opened, a considerable amount of thin serosanguineous fluid escaped. Immediately beneath the incision a large, firm, brownish-red mass pre-

48. This patient was presented before the Surgical Section, New York Academy of Medicine, on May 7, 1926.

sented and seemed to fill the entire right side of the abdomen. On exploration this mass proved to be made up of the entire great omentum, which was twisted on itself just below the right half of the transverse colon where the twisted portion was gathered into a narrow pedicle with seven turns. Distal to this pedicle was the large, firm, beef-red mass representing the entire great omentum. The appendix was acutely inflamed, but was not involved in the omental mass. The omental pedicle was tied off, and the mass was removed. The appendix was then removed in the usual manner, and the abdomen was closed without drainage. Convalescence was uncomplicated, and the patient subsequently enjoyed perfect health.

The pathologic report was: subsiding acute appendicitis and hemorrhagic infarction of the great omentum.

CASE 2.—M. O., a white man, aged 38, a clerk, was admitted to the fourth surgical division, Bellevue Hospital, on Feb. 9, 1928. He had enjoyed excellent health until the day before admission, when he first noted a dull pain in the right lower abdominal quadrant. He was able to continue his work and take food without distress, but the pain grew steadily worse during the day. On going to bed that night, he found that he could not lie on the affected side without great discomfort. At no time was there any associated nausea, vomiting, diarrhea or urinary disturbance. No history of trauma or physical strain was obtained. The bowels had been regular up to the onset of the present attack. During the night the pain became so severe that the patient decided to enter the hospital.

Examination disclosed an unusually robust white man whose weight was estimated at about 200 pounds. General physical observations were entirely negative, except for the protuberant obese abdomen which was distended and tympanitic. The right rectus muscle presented some spasm, and there was definite tenderness localized in the region of McBurney's point. There was a suggestion of a mass in this region, but the muscular spasm and obesity rendered this uncertain. The rectal temperature on the patient's admission was 101 F.; urinalysis yielded negative results; the white blood cells numbered 11,600, with 70 per cent polymorphonuclears. The preoperative diagnosis was acute appendicitis.

Operation.—A right rectus muscle splitting incision was made. A small amount of thin, bloody fluid was evacuated on opening the peritoneum. Just beneath the incision, a narrow tongue of omentum was found extending downward and outward to the right to become adherent to the anterior abdominal wall in the region of McBurney's point. Near its distal end a twist had taken place, shutting off a tab the size of a walnut, which was black and gangrenous. The appendix and gallbladder were normal in appearance. The mass was separated from the abdominal wall, and the tongue of omentum was tied off and amputated at its origin. The abdomen was closed without drainage. Recovery was uneventful, and the patient remained free from symptoms.

Pathologic examination revealed a mass, 3 by 2 by 1 cm., which resembled thyroid tissue. A section showed fat tissue infiltrated by hemorrhage.

Comment.—Cases 1 and 2 are typical examples of two varieties of secondary torsion. Case 1 belongs to the unipolar type referred to under group *a* as associated with an extra-omental inflammatory focus—in this instance acute appendicitis. It illustrates well the tendency of this type of unipolar torsion to form large masses with thin, tight pedicles and a well advanced stage of circulatory impairment. In this

instance the mass filled the entire right side of the abdomen and measured 12 by 5 by 3 inches (30 by 12 by 7 cm.) (fig. 2). The history, course, physical and laboratory observations simulated the classic picture of acute appendicitis so accurately that a differential diagnosis could not be made preoperatively.

Case 2 belongs to the bipolar variety of torsion referred to under group *c*. Adhesion of the free margin of an omental process to the anterior abdominal wall provided the "second fixed point," and rotation had taken place between this point and the normal fixed point at the origin of the omental tongue. In this type of torsion the mass is characteristically small, and there may be two or more such masses with intervening pedicles. The symptoms suggested a diagnosis of appendicitis, but in this instance the picture was not as characteristic as that in case 1, and might well have been questioned. Both these patients had the stature that is so frequently associated with torsion of the omentum that its etiologic significance must be given some consideration, namely obesity, an excessive deposit of subcutaneous fat and a lax, protuberant abdomen. In both instances the patient had led a sedentary life, there was no history of abdominal trauma or physical strain, and there was no evidence of previous similar attacks.

CHRONIC RECURRING TORSION

In discussing the foregoing classification, the suggestion was made, purely on theoretical grounds, that temporary twists of the great omentum occur fairly frequently, owing to its character and surroundings. It was assumed that such twists were incomplete in the sense that they did not produce permanent circulatory impairment, but that they were expressed clinically by certain vague abdominal symptoms. It was further shown, in analyzing the histories of the various types of acute torsion, that in a definite percentage of these cases the immediate acute attack of torsion (complete) had been preceded, over considerable periods of time, by intermittent subacute manifestations of a type conceived of as incomplete in origin.

In their consideration of omental torsion, Corner and Pinches [9] were impressed by the significance of these antecedent subacute disturbances, and in the conclusion of their discussion they stated:

> These (the previous attacks) may be produced by colic, appendicitis or some such condition quite unconnected with omental twists. But many twists—sometimes 10—are frequently found. These have been formed by slow increments of the torsion. It is tempting and not irrational to refer the previous attacks of pain to some of the increments being greater or more forcible than the others. This is borne out with regard to the testis; for instance, the more acute increase in torsion which caused the attack of pain has been untwisted giving perfect relief. Future observers will gain much in paying more attention to the history

of cases of this nature. If this surmise is correct, that these attacks or some of them are produced by increased twists although only the more severe cases are yet recognized, the previous history of the patients illustrates the clinical history of milder examples due to subacute and chronic torsion of the omentum and hitherto unrecognized. It may be expected confidently that the observations in the future will tend to make occurrence of omental torsion more frequent just as in cases of torsion of the bowel and of the testis.

The clinical effects of this type of temporary torsion are seen in structures other than the omentum in which a comparatively large, dense body is suspended by a long, thin pedicle; for example, it is a matter of common clinical experience that temporary incomplete twists of the pedicle of an ovarian cyst may give rise to repeated transitory abdominal attacks the nature of which is unrecognized until the final decisive complete attack leads to operation and discloses the source of the preceding symptoms. I recently saw a premature infant 7 months of age, in whom a swelling in the right groin and scrotum had suddenly developed. For several weeks previous to this occurrence, the patient had had frequent attacks of colic; as there was no associated gastric or intestinal disturbance, the attending pediatrician was at a loss to account for these attacks. At operation, carried out under the diagnosis of strangulated hernia, it was found that a torsion of the right spermatic cord existed and that gangrenous changes in the testicle required its removal. In the light of the observations at operation, it seems logical to refer the antecedent attacks of colic to transitory, incomplete twists of the spermatic cord which had spontaneously untwisted themselves before complete circulatory obstruction supervened.

From the foregoing considerations, certain facts may be assembled in support of the contention that transitory incomplete twists occur in the omentum, and that such twists are responsible for obscure recurrent abdominal symptoms the nature of which usually is unrecognized: 1. It is well known that transitory twists of this type occur in structures other than the omentum (ovarian cysts, testicle) and give rise to similar recurrent abdominal attacks. 2. Physical factors analogous to those favoring torsion in these structures can be demonstarted in the omentum. 3. In a considerable percentage of the cases of acute torsion in this series, there is evidence that the final acute attack was antedated by repeated transitory abdominal disturbances, which may be interpreted as due to incomplete omental twists.

This conception of the omentum as a source of obscure, recurrent abdominal symptoms opens up interesting clinical possibilities, and the inclusion of these conditions under the separate group of incomplete torsion represents an effort to emphasize these possibilities as well as to distinguish them from the acute complete types of torsion.

Abdominal diagnosis is far from precise in spite of the development of exact methods of investigation. Everyday clinical experience is rich in instances of obscure abdominal disturbances the character of which cannot be definitely decided without resort to "exploratory laparotomy" and the frequent failure of this procedure to demonstrate a convincing pathologic process as well as to relieve symptoms is too well known. Undoubtedly, chronic infections of the appendix, gallbladder, stomach and pancreas are factors in many or most of these instances. If it is granted, however, that transitory incomplete twists of the omentum occur, and that these twists are responsible for the clinical manifestations already referred to, it is tempting to attribute to this cause at least a small percentage of these cases.

Systematic examination of the omentum is rarely carried out during the course of exploratory operations, because it is not often thought of as a possible source of trouble. A pathologic process capable of initiating omental twists (e. g., a localized area of fibrosis) may therefore be easily overlooked or its significance may not be appreciated.

The following case illustrates the typical pathologic and clinical features of incomplete torsion of the chronic recurring type:

CASE 3.—G. H., a man, aged 51, a valet, was admitted to the Postgraduate Hospital on Nov. 7, 1929, complaining of pain in the lower part of the abdomen. His present trouble began two months before admission to the hospital, when he began to have periodic attacks of dull pain across the lower part of the abdomen, which was most marked in the suprapubic region. These attacks usually lasted two or three days and were always associated with indigestion and occasionally with vomiting. The movements of the bowels were regular. There was no diarrhea, dysuria or polyuria and no radiation of pain. Between attacks the patient was completely free from symptoms, appetite was normal and there was no indigestion or loss of weight. A large right inguinoscrotal hernia was present but had never given trouble. It had always been reducible and he had never worn a truss. During his most recent attack, he thought that the hernia had increased in size. His previous health had always been good, with the exception of an attack of acute appendicitis in 1926, during which I removed his appendix.

Examination disclosed a large, robust man, well nourished and well developed. General examination gave negative results except that the abdomen was rather protuberant and presented a right paramedian scar. There was a large right scrotal hernia which was painless and easily reducible. It apparently contained intestine. There was no muscular rigidity, and no mass or free fluid could be detected. The temperature and blood count were normal; the blood pressure was 120 systolic and 80 diastolic. The urine showed only a faint trace of albumin.

Operation.—A right inguinal incision was made, and a large, thick inguinoscrotal sac containing only small intestine was delivered. The cord and vessels were dissected free, the sac was opened and the contents reduced. There was no omentum in the sac. The neck of the sac at the internal ring was tremendously dilated and easily permitted abdominal exploration. A large mass of fat-infiltrated omentum was gathered in the right side of the abdomen and hung down into the pelvis, but was not adherent at any point. This mass of omentum was delivered through the internal ring and its redundancy was such that it reached well below the

lower angle of the incision. At the most dependent point of the free margin of the omentum, there was a circumscribed area of thickening and fibrosis forming a mass about the shape and size of a small orange (fig. 3). This thickened area seemed to form the apex of the omental mass, and the lateral margins seemed to drape themselves naturally about the axis thus formed. There was no evidence of active twisting or pedicle formation. The whole mass assumed an elongated ropelike shape at the end of which hung the "omental ball"; the resulting relations might be likened to a handkerchief suspended by one corner and a weight fixed to a diagonally opposite corner while the lateral free corners fall loosely about the central axis. The omentum was drawn down as far as possible, ligated at the highest accessible point and amputated distal to the ligatures. The abdominal cavity could now be easily and thoroughly explored through the enormously dilated

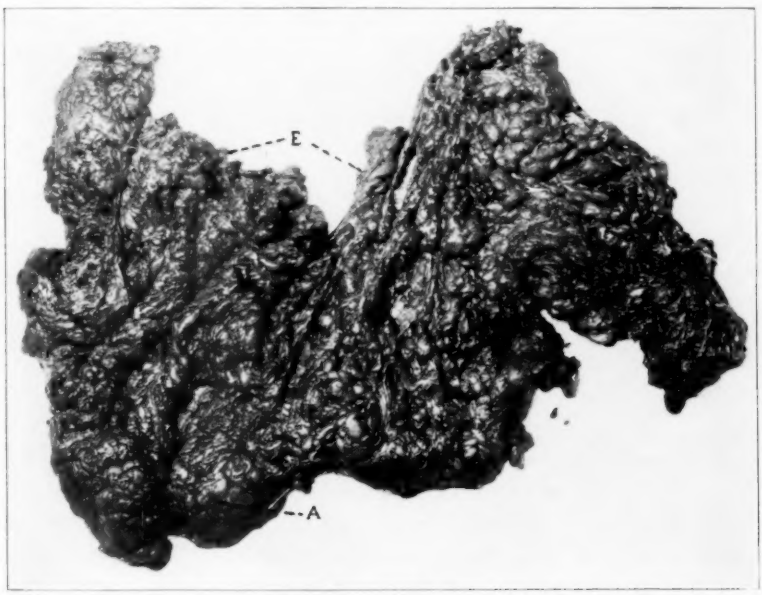

Fig. 3.—Specimen removed from patient in case 3, showing mass of redundant normal omentum with omental ball (*A*) at dependent point. *E* indicates cut edge of omentum.

internal ring. Careful palpation of the stomach, gallbladder, large intestine and pelvis failed to disclose any other pathologic process. The hernial sac was removed, its neck tied off and the canal repaired by the Bassini method.

The patient made a good recovery. When last observed, he had been under constant observation for six months. The abdominal symptoms, which were recurring at weekly intervals previous to operation, had completely disappeared, and the patient was enjoying perfect health.

Comment.—This case is presented as an example of incomplete torsion of the omentum of the chronic recurring type for the following reasons: 1. The pathologic and mechanical conditions found in the omentum favored the development of transitory twists, although

no actual twist or pedicle formation was present at the time of operation. 2. The recurrent abdominal symptoms experienced by the patient were not unlike those reported as preliminary manifestations in a large percentage of the cases of acute torsion in this series, and in latter instances, accepted as evidence of incomplete twists which freed themselves spontaneously. 3. The attacks were completely cured by removal of the offending omentum.

The omental conditions described, i.e., an elongated ropelike structure suspending a comparatively large, dense, tumor mass, seems to provide the mechanical conditions necessary for the development of transitory twists, and it is clear from experience with acute complete torsion that, when such conditions exist, the added stimulus of a blow, a fall or a sudden change of position is all that is required to initiate the twisting process.

There were only two other instances in the series—cases reported by Swain [49] in 1920—in which the clinical manifestations and associated pathologic process seemed to justify inclusion under the classification of incomplete torsion. The first was in a man, aged 58, with pain in the upper right abdominal quadrant, a palpable mass, tenderness and elevation in temperature. At operation a congested mass of omentum was found near the hepatic flexure, but there was no evidence of a pedicle or twist. The second case was in a woman, aged 25, with vague general abdominal pains which eventually localized about the umbilicus and in the lower right abdominal quadrant, where a palpable tender mass was detected. Operation disclosed a congested mass of omentum without twists or pedicle formation. In both cases the pathologic process was described as "active hyperemia of the omentum," and the subsequent course in both indicated that the process bore a causative relation to the symptoms produced.

Although the number of clinical instances of incomplete torsion is not impressively large, the available data at least serve the purpose for which this group was primarily introduced into the classification, namely the demonstration of the reality of incomplete omental twists and their potentiality for producing abdominal symptoms. Taking into consideration the evidence presented in case 3, together with that derived from experiences with complete omental torsion and torsion in other structures, I would summarize the salient facts concerning incomplete torsion in the following conclusions:

1. Incomplete twists of the omentum may, under certain conditions, occur and recur intermittently without progressing to the stage of the complete circulatory impairment seen in complete torsion. 2. These incomplete twists are accompanied, as a rule, by recurrent, subacute

49. Swain: Bristol Med.-Chir. J. **37**:202 (Dec.) 1920.

abdominal symptoms which, in the light of present clinical experience, are usually attributed to chronic disturbances of the appendix, stomach or gallbladder. 3. The localized areas of omental thickening or fibrosis which characterize incomplete torsion may be readily overlooked during the course of an exploratory laparotomy or, if encountered, their true significance may not be appreciated. It therefore appears probable that incomplete omental twists are responsible for obscure abdominal symptoms more frequently than the foregoing statistics indicate.

DIAGNOSIS

Considering the pathology and clinical picture presented by torsion of the omentum, it is not surprising to find that preoperative diagnosis

TABLE 3.—*Preoperative Diagnosis in 138 Cases of Torsion of the Omentum*

Diagnosis	Torsion With Hernia	Torsion Without Hernia			Total	
		Primary	Secondary	Incomplete Torsion		
Acute appendicitis	24	24	16	1	65	82
Appendiceal abscess	5	3	3	..	11	
Appendicitis with peritonitis	1	1	..	..	2	
Appendicitis (subacute)	..	3	1	..	4	
Strangulated inguinal hernia	15	..	..	..	15	
Inguinal hernia	3	..	..	..	3	
Cholecystitis	1	4	3	1	9	
Torsion of ovarian cyst	3	1	3	..	7	
Intestinal obstruction	1	1	1	..	3	
Torsion of omentum	8	1	1	1	11	
Omental hernia	1	..	..	..	1	
Tuberculosis of cecum	1	..	..	..	1	
Strangulated ventral hernia	3	..	..	..	3	
Torsion of uterine fibroma	..	..	1	..	1	
Torsion of spermatic cord	1	..	..	..	1	
Pelvic tumor	..	..	1	..	1	

of this condition has been both varied and inaccurate. Of the entire series of 164 cases, a preoperative diagnosis was recorded in 138. In table 3 is tabulated a list of these diagnoses apportioned to the type of torsion in which they were made.

Analysis of the figures in table 3 brings out points of practical interest; for example, in eighty-two (59 per cent) of the cases, a diagnosis of acute appendicitis or acute appendicitis with abscess or peritonitis was made. This diagnosis was almost twice as common among the pure abdominal types of torsion (torsion without hernia) as in the type associated with herina (torsion with hernia). Of the nine diagnoses of involvement of the gallbladder, seven were made in the pure abdominal type of torsion, while the twenty-one diagnoses of hernia, strangulated or incarcerated, were made in the hernial type. In only eleven (7.9 per cent) cases was a correct diagnosis of torsion of the omentum recorded and eight (72 per cent) of these showed the hernial type of torsion.

From these figures it appears that torsion of the omentum, whether of the hernial or the purely abdominal type, simulates closely the clinical manifestations of acute appendicitis and, to a lesser degree, of cholecystitis. It is furthermore evident that the associated hernia in the hernial types is an important factor in diagnosis since 72 per cent of the correct diagnoses were made in cases of the latter type, while the diagnosis of acute appendicitis was made twice as frequently in the nonhernial or pure abdominal types.

Smythe [20] studied seven cases of pure abdominal torsion in six of which the diagnosis of acute appendicitis had been made; despite these discouraging figures he proposed an elaborate system of differential diagnosis which is summarized briefly in table 4.

TABLE 4.—*Differential Diagnosis of Acute Appendicitis and Torsion of the Omentum (Smythe)*

	Torsion of the Omentum	Acute Appendicitis
Age	Usually between 35 and 55	Usually between 15 and 25
Sex	Practically all cases in males	Males predominate 6:1
Pain	Right lower quadrant in 85%; not sudden or violent	General, then limited to right lower quadrant; may be acute or violent
Pulse	Not much affected	Usually accelerated (100 to 140)
Temperature	99 to 100 F.	101 to 103 F.
Mass	Early, diffuse, large	Second or third day; circumscribed
Rigidity	Not marked	Marked
Percussion	Dulness	Resonance
Leukocytosis	Usually absent	Usually present
Nausea and vomiting	Uncommon	Common

In commenting on such a method of differential diagnosis, it is necessary to point out only that it represents an overemphasis on isolated symptoms and that its assumptions, based on a limited number of cases, are not borne out by the symptom analysis of my larger series; for example, it was previously shown that nausea and vomitting, far from being "uncommon" in torsion, occurred as early symptoms in 45 per cent of the cases. Similarly elevation of temperature was present in 58 per cent and acceleration of pulse rate in 26 per cent of this series. It is doubtful if a similar series of cases of acute appendicitis will show any material variation in the incidence of these important diagnostic criteria. So far as the blood picture is concerned, it must be admitted that leukocytosis as an isolated symptom in acute appendicitis is often unreliable and that to place too great dependence on it is to court disaster. Furthermore, this symptom is not "usually absent" in torsion, for the present series discloses white cell counts varying from 12,000

to 19,000 in an appreciable number of cases. Finally, it is self-evident that youth is no bar to torsion or old age to appendicitis.

Clinical symptoms, such as these, can be properly evaluated only when it is recognized that they are nonspecific reactions of a definite physiologic mechanism to varying types of pathology. The symptom of nausea and vomiting, for instance, must be regarded as an expression of reflex sympathetic irritation in which the mechanism and results are identical regardless of the character, but proportional to the intensity, of that irritation. It is therefore to be anticipated that this symptom will occur with approximately equal frequency in both torsion of the omentum and acute appendicitis, provided these lesions are of the same degree of severity. Elevation of temperature, pain, acceleration of pulse rate, etc., obey the same rule and merit the same consideration.

Analysis of the cases in this series has brought out, however, characteristics that may be taken as specific for torsion of the omentum, and that should, in consequence, be helpful in preoperative diagnosis. For example, analysis of the cases has shown that a significantly large percentage of patients with torsion are of a vigorous, robust or obese type whose occupation or environment subjects them to frequent sudden and severe physical strains and abdominal trauma. In the three cases that I observed, the patients were strikingly obese, weighed about 200 pounds (90.7 Kg.) and presented excessive deposits of subcutaneous fat, justifying the recognition of a "torsion habitus." These factors, torsion habitus and physical strain or trauma, are of undoubted importance in the predisposition to torsion of the omentum.

In persons of this type, especially with an antecedent history of repeated attacks of vague, subacute, abdominal symptoms, the acute onset of severe pain in the right lower quadrant of the abdomen, accompanied by the rapid development of a large abdominal mass and free fluid, presents a suggestive picture. The mass itself has certain characteristic features: It is rapid in development, remarkebly large, often freely movable and frequently painless. Nausea, vomiting, elevation in temperature, leukocytosis, etc., are merely incidental to a well established peritoneal irritation.

When the hernial types of torsion are concerned, diagnosis is simplified, to a considerable degree, by the presence of the hernia which is always an important clue. This hernia is typically of a long-standing, painless, scrotal variety, the contents of which are almost invariably omentum and have always been easily reducible. When a hernia of this type suddenly changes its character to become irreducible, painful and enlarged and when a large, rapidly developing mass appears simultaneously above Poupart's ligament, acute torsion of the combined hernial type is to be suspected and treated accordingly.

Fig. 4.—Section of omentum from case 1, showing vascular thrombosis, extensive interstitial extravasation and hemorrhage; × 60.

PATHOLOGY

The gross pathologic changes characteristic of the various types of torsion are well illustrated by cases 1, 2 and 3, and these have already been given in detail. Case 1 is an excellent example of the pure abdominal type of acute torsion without hernia (primary or unipolar variety). The twist had taken place at the transverse colon, and the entire omentum was involved in a firm, beef-red, edematous, congested

Fig. 5.—Section of omentum from case 2, showing marked thrombosis, hemorrhage and extravasation; reduced from × 60.

mass filling the right side of the abdomen (fig. 2). The microscopic changes observed on section of this mass were disappointing. The smaller and larger vessels showed evidence of stasis and consequent thrombosis, while there was extensive interstitial extravasation of red and white blood cells with areas of actual hemorrhage, particularly marked about the margins of the mass. The actual structural changes were, however, surprisingly limited, and there was little evidence of an inflammatory process or organization (fig. 4). In case 2, an example

of acute intra-abdominal torsion without hernia (secondary or bipolar variety), the mass was small but the hemorrhagic and thrombotic features seemed grossly more pronounced. Yet here also the microscopic details, with the exception of more extensive hemorrhage, were similar to those in case 1 (fig. 5).

Case 3, an example of incomplete torsion, of the chronic recurring variety, presented grossly and microscopically a picture which contrasted markedly with that of complete, acute torsion. The gross omental

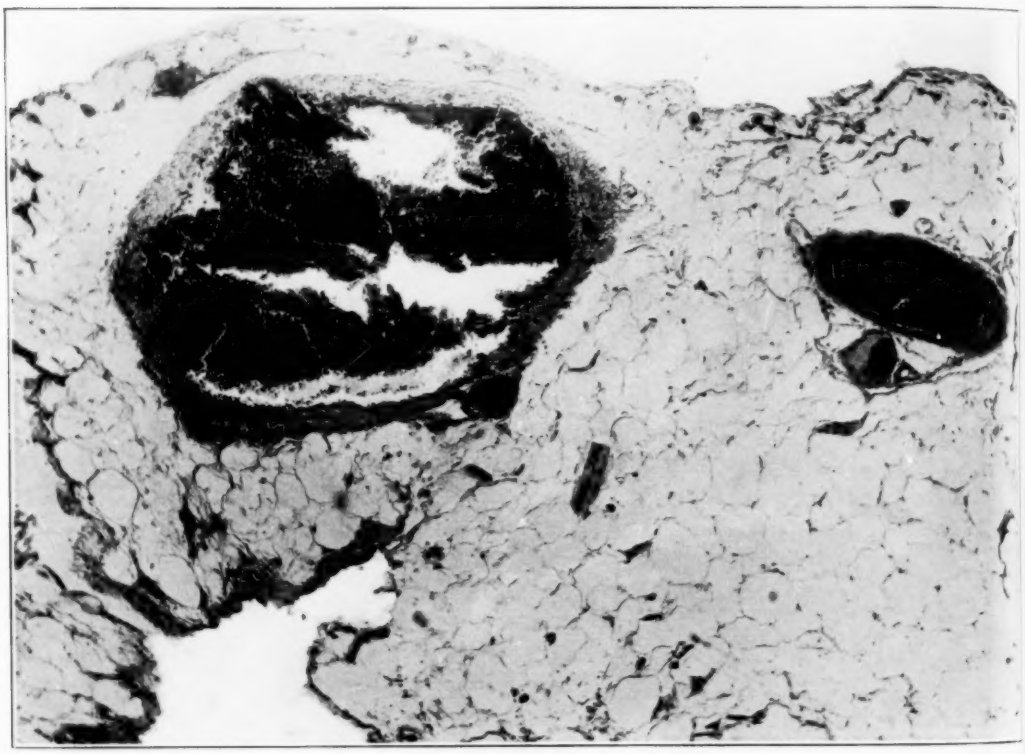

Fig. 6.—Section through omental ball from omentum in case 3 showing marginal endothelial hyperplasia with little hemorrhage or extravasation; reduced from × 60.

changes comprised a circumscribed, knobby area of thickening at the dependent margin of the omentum (fig. 3). The red, congested edematous appearance so characteristic of complete torsion was not in evidence, the natural color of the omentum being preserved, and the sole departure from normal seemed to be one of consistency only. The microscopic section of this specimen also showed a distinctively different picture in that there was a striking absence of interstitial

extravasation and hemorrhage although there was some indication of vascular stasis and early thrombosis. There may be noted, however, in this section a characteristic thickening and endothelial hyperplasia along the margins of the mass (fig. 6). This is then a definite organic structural change which has not been observed in the acute complete types and which is no doubt to be interpreted as the result of repeated mild irritations to which the latter types are not exposed.

ETIOLOGY

In considering the etiology of omental torsion, it is of primary importance to determine whether or not the causative factors are intrinsic or extrinsic. Does the omentum itself possess the power, under certain conditions, to initiate the twisting process; is this process dependent on forces exerted by adjacent structures, or are both these influences concerned in producing the conditions observed?

It is interesting to note that although the available experimental evidence is conservative, tradition has been generous in attributing to the great omentum a great variety of qualities and functions. Tait called it "the arbiter of peritoneal tides," while Robinson is responsible for the appellation "policeman of the belly." Among the older writers all manner of bizarre virtues were given to it. Heusner [50] summarized the views held up to the present as follows:

(1) An apron protecting the intestines from cold (Aristotle).

(2) A fixation ligament for the transverse colon (Versalius).

(3) Protects abdominal viscera from friction (Verboyen).

(4) Draws filled stomach downward, facilitating respiration (Hansen).

(5) An organ separating and moistening the viscera, as well as an organ of absorption.

(6) An expanded lymph gland exercising the functions of the hemolymph tissues of the abdomen.

(7) A protective and defensive agent in various pathologic conditions and accidents.

According to the most modern conceptions, based on extensive clinical observations, the omentum is endowed with a spontaneous ameba-like movement—an "intelligent chemotaxis"—through which it exercises the power to plug openings in a ruptured viscus, limit inflammatory processes and even act as an hemostatic agent in cases of intraperitoneal hemorrhage.

The existence of an intrinsic "intelligent chemotaxis" in the great omentum was, however, called into question by the experimental studies of Rubin,[51] who concluded that the sole functions of this structure

50. Heusner: München. med. Wchnschr. **52**:1130, 1924.

51. Rubin: Surg., Gynec. & Obst. **12**:117, 1911.

are: (1) to afford fixation for the intestine; (2) to provide freedom of intestinal movement, and (3) to serve as a vehicle for the arteries, veins and nerves. These studies suggest that the omentum does not invariably repair defects, that whatever protective power is displayed depends on its properties as peritoneum and that its usefulness in inflammatory lesions is due to its power(*a*) to form adhesions isolating and rendering innocuous the effects of inflammation and (*b*) to absorb and eliminate the toxic products. So far as "intelligent chemotaxis" is concerned, the displacements of the omentum are explained as due to (1) intestinal peristalsis, (2) intra-abdominal tensions, (3) the suction action of the diaphragm and (4) distention of the stomach and colon by gas or fluid. In the presence of free intraperitoneal fluid the omentum is invariably curled up on its surface and is usually found on the anterior face of the liver when there is a large amount of fluid. For the purposes of the present discussion, therefore, the net result of these experiments serves to discredit any intrinsic power of the great omentum to initiate the twisting movement through which the condition of torsion is brought about. Extraneous influences must be sought in explanation of the phenomenon of torsion, which is to say that this condition presupposes the presence of a concomitant intra-abdominal pathologic process.

In seeking to establish the nature of the extraneous factors leading to omental torsion, it is of importance to point out that the great omentum normally presents certain structural characteristics which have a contributory influence. This structure varies in length from a few inches to an enormous elongated mass reaching deep into the pelvis. Meckel is authority for the statement that it passes into the pelvis in 25 per cent of males and in 50 per cent of females. The right border is longer and much more mobile than the left, which is usually found tucked up in the left hypochondrium. In addition, the right side is frequently distinguished by the presence of two or more tongue-like processes; this fact, together with the greater length and mobility on the right side, is significant in view of the prevalence of torsion on this side. General body nutrition is accurately reflected by the omentum; in emaciated persons it is thin and shriveled, while in those who are well nourished or obese there is an early and rapid accumulation of fat in the organ which may attain tremendous weight, size and thickening. In this connection the discussion of a "torsion habitus" is to be recalled. Finally it was shown by Payr, who carried out experimental studies to determine the causes of torsion, that the disposal of the omental circulation was such as to favor the twisting process. According to this observer, the omental veins are unusually long and tortuous and when, for any reason, they become engorged there is a tendency toward their spiral rotation about the shorter rigid artery acting as a mesentery, so that the first step of torsion may thus be initiated.

Pathologic changes in the omentum and in its environment constitute the actual causative factors of torsion, but these factors are not necessarily identical for all the recognized varieties of torsion. For example, the mechanics of torsion when a weight is suspended free at the end of a string (unipolar torsion) are quite different from the conditions that obtain when a handkerchief is suspended by diagonally opposite corners with freedom of rotation between these fixed points (bipolar torsion).

In principle, conditions favorable for unipolar torsion are presented by any local increase in omental density capable of producing a narrowing and lengthening of the omentum until it is suspended by a true pedicle. There is then lacking only the proper stimulus to initiate the twisting process. The statement has been made by Baldwin [52] that all that is necessary for this type of torsion is the existence of a pedunculated omental tumor with no mechanical obstacles to rotation.

Concerning this omental tumor mass, three distinct types and manners of origin may be distinguished: (1) It may be a true tumor of a neoplastic variety; (2) it may be due to an inflammatory fibrosis secondary to an acute process in an adjacent structure, e.g., the appendix, and (3) it may be produced by the mechanical forces of an omental hernia.

Of the true omental tumors, the embryonal lymphangioma, a cystic growth encountered in early life, appears to be most frequently associated with torsion. According to Fink,[53] thirty-eight of these tumors had been reported up to 1914, and in the present series there were three instances of torsion due to this type of tumor. Prutz [54] found that torsion occurred with sarcoma, secondary carcinoma and echinococcus cysts. In a case reported by Owen,[55] a hydatid cyst had produced a definite torsion of the omentum with multiple twists of the pedicle.

Inflammatory tumors of the omentum are produced by secondary extension from an adjacent focus, e.g., the appendix. These tumors persist in the omentum as localized areas of fibrosis long after the original focus has subsided or has been removed and may thus become the starting point for a torsion of the omentum. My case 3 was an instance of this sequence of events.

Four years previous to the onset of the symptoms of torsion, this patient, a large obese man, was operated on for acute appendicitis requiring drainage. At the second operation for torsion a characteristic localized area of fibrosis was identified as responsible for the twisting pro-

52. Baldwin: Prag. med. Wchnschr. **35**:559, 1910.
53. Fink: Surg., Gynec. & Obst. **15**:70, 1914.
54. Prutz: Deutsche Ztschr. f. Chir. **46**:280, 1913.
55. Owen: Intercolon. M. J. Australasia **2**:459, 1921.

cess. It appears logical to regard this omental tumor as a secondary manifestation of the antecedent appendical inflammation. In my case 1, acute complete omental torsion was found to be associated with definite evidence of subsiding acute inflammation of the appendix. Analysis of the cases in this series discloses thirteen other instances in which some form of acute or subacute lesion of the appendix either preceded or was coexistent with the torsion. These cases were all classified as torsion secondary to inflammatory tumors of the omentum on the assumption that an infective process adjacent to the omentum had induced a localized reactive fibrosis which, in turn, accounted for the progressive steps leading to torsion. It seems probable that this sequence of events obtains more frequently than the foregoing figures indicate and that other organs, such as the gallbladder, stomach or pelvic structures, may, in a similar manner, set up localized reactive omental tumors which persist, to cause trouble, long after the original causative focus has ceased to exist.

The third, and undoubtedly the most common, source of localized omental fibrosis and pedicle formation is the long-standing inguinal hernia. It is a well recognized clinical fact that these herniae bring about an actual lengthening of the omentum, a portion of which eventually comes to lie in the scrotum. According to Prutz, the portion of the omentum in the scrotum is exposed to comparatively little pressure while that in the inguinal canal is compressed into a roll or cord which gradually becomes thinner as the scrotal portion becomes edematous and thickened in consequence of the compression above. The resulting tumor mass suspended by its long thin pedicle now presents conditions ideal for unipolar torsion if the proper initial twisting impulse is added. Repeated attempts at reduction of this mass frequently supply this necessary impulse. The movement of the omental mass back and forth through the unequally calibrated inguinal canal has been compared by Hadda to the rotation of the fetal head passing through the parturient canal or to a bullet forced through the bore of a pistol barrel. Attempts of the rigid, fibrosed tumor to adjust itself to the varying diameters of the inguinal canal impart a rotatory motion to the advancing tumor, and this turning movement is accentuated by the fibers of the transversalis and oblique muscles. These twisting movements, however mild, lead progressively to further edema and fibrosis of the tumor, the increasing size and weight of which accentuate the degree of twist; a vicious circle is thus produced, which determines the final stages of complete torsion.

In the pure abdominal, unipolar types of torsion, Hadda's theory of the initial twisting impulse is obviously not applicable. It has been said of this type that, given a pedicled tumor, all that is necessary for

torsion is the absence of mechanical obstacles to rotation. Nevertheless it is evident from a study of certain histories that definite factors play a part in initiating this rotation when the proper relation of pedicle and tumor exists. For instance, there were thirty-one cases of abdominal torsion in the series in which the onset of acute manifestations coincided strikingly with such occurrences as blows on the abdomen, violent coughing spells, horseback riding, dancing, being violently whirled about while wrestling, stooping over a tub and various other sudden physical efforts. In these cases a preexisting pedicled tumor of the omentum is assumed, "with no mechanical obstacles to rotation"; under these conditions it is entirely conceivable that such a combination of events is competent to precipitate the rotation necessary to determine a complete torsion. Prutz thought that a change in the position of the entire body was sufficient, under the influence of gravity, to supply the necessary impulse. In the cases reported by Morestin and Taddei, torsion was attributed to violent peristalsis induced by drastic catharsis. Kraske,[56] observed a case in which omental torsion was said to be due to the failure to rearrange the omentum after a protracted operation in the Trendelenburg position. While the relationship of some of these factors to torsion many well be questioned, it is nevertheless fairly definitely established that, given a free, pedicled omental tumor, a number and variety of forces enter into the production of the initial rotatory movement.

As has been noted, bipolar torsion presents mechanical conditions quite different from those of unipolar torsion, and those conditions favorable for the former type are established when a tongue of the free margin of the omentum becomes fixed or adherent to some adjacent intra-abdominal structure. This abdominal point of fixation is, in the majority of instances, the result of a localized inflammation in the structure to which the omental tip becomes attached. The appendix, gallbladder, ovaries and tubes play the largest part in this process, but infrequently fixation is noted at the uterus, right kidney, ascending colon and stomach. Formation of the second fixed point by purely mechanical means is suggested by the experience of Luckett who encountered a bipolar torsion in a patient operated on ten years before for right inguinal hernia. The twisted tongue of omentum in this case was adherent to the old hernial cicatrix, and the author concluded that the original operator had included a tongue of omentum within the ligature used to tie off the hernial sac. This experience in particular and the principle of bipolar torsion in general suggest that the deliberate fixation of a portion of the omentum by suture to an adjacent organ is not without danger, and that the accidental fixation which may occur during closure of laparotomy wounds is to be avoided carefully.

56. Kraske, quoted by Aimes (footnote 5).

Given the two fixed points of bipolar torsion, there is still to be determined the character of the impulse which initiates rotation about the axis thus formed. Payr and Frederick observed cases of bipolar torsion in which the omental tip was adherent to an ovarian cyst. Primary torsion developing in the latter was thus transmitted to the omentum causing a secondary bipolar torsion therein. This type of causation is, however, obviously infrequent, and it is entirely probable that the exciting factors of rotation in bipolar torsion are not unlike those described for unipolar torsion, viz., changes in the position of the body, blows on the abdomen, sudden severe physical strains, hyperperistalsis of the intestine, etc.

CONCLUSIONS

Marchette reported the first case of torsion of the omentum in 1851. Corner and Pinches, in 1905, collected 54 cases, which included 3 of their own. A careful search of the literature from that date to 1929 yielded 164 additional cases, to which I add 3 personally observed cases for a total of 217. It has been my purpose in this report to assemble the clinical and pathologic data presented by this group and on that basis to evaluate the clinical importance of torsion of the omentum. As a result of this study, it is possible to summarize briefly the salient features of this condition in the following conclusions:

1. The normal environment and anatomic arrangement of the great omentum are such as to favor rotation of this structure about its long axis, while the introduction of certain mechanical conditions and pathologic changes within and adjacent to it supply the immediate exciting causes of such rotation.

2. It is clearly established that omental twists are capable of producing clinical symptoms of an acute and chronic character, and that this condition therefore merits some consideration in the diagnosis of abdominal conditions.

3. Variation in the intensity of degree of rotation determines two basic clinical and anatomic types of torsion: (1) the complete, in which acute, progressive symptoms and marked pathologic changes are dependent on complete permanent circulatory obstruction at the site of twist, and (2) the incomplete, which is characterized by vague, chronic recurring symptoms and mild pathologic changes in consequence of incomplete, partial or temporary obstruction which permits repeated spontaneous restitution before pronounced organic changes develop.

4. Diagnosis presents obvious difficulties as indicated by the fact that a correct preoperative diagnosis was recorded in only 7.9 per cent of 217 cases. A statistical study of the series, however, emphasizes

certain essential clinical manifestations and points of history, the recognition of which should enhance the precision of a preoperative diagnosis.

5. The deliberate fixation of the free margin of the omentum by suture to an adjacent structure may be the source of future complications, and its accidental inclusion during abdominal closure is to be avoided carefully.

Fig. 7 (case 4).—Photograph showing the narrow tight pedicle and the distal gangrenous mass of the omentum.

NOTE.—The following case of omental torsion was encountered shortly after the submission of this article for publication.

CASE 4.—G. W., aged 47, a barber, was admitted to the fourth surgical division, Bellevue Hospital, on April 1, 1931, with a condition diagnosed as acute appendicitis. Three days before admission he had noted a dull pain in the right lower quadrant of the abdomen. The pain was constant, did not radiate and was not accompanied by nausea, vomiting, diarrhea or urinary disturbances. The appetite had remained good, although the patient had been markedly constipated during the week preceding the attack. He had never experienced similar attacks, and the previous history was unimportant.

Examination disclosed a robust, obese man who weighed 190 pounds (86.2 Kg.). General physical examination gave negative results. The abdomen was protuberant and lax. There was marked, acute, localized tenderness over McBurney's point, but no muscular spasm could be detected. In this same region a small, hard, painless and freely movable mass was palpable. There was no gross evidence of free fluid. The temperature was 100.6 F.; the pulse rate, 100. There were 9,550 white blood cells, with 72 per cent polymorphonuclears. Urinalysis gave negative results. A diagnosis of acute appendicitis was made, and immediate operation was advised.

Operation.—When the peritoneum was opened, a small amount of free serosanguineous fluid was found. The appendix was decidedly injected, and the gross appearance justified a diagnosis of subacute appendicitis. Exploration was extended, however, and resulted in the finding of a hard mass above and to the right of the incision. On delivery, this proved to be a bluish triangular mass of omentum about the size of a human hand. Its pedicle was thin and tightly constricted and could not be untwisted. The mass swung free in the peritoneal cavity; it was not attached to any other structure, and it was apparently not associated with the appendix. There was no inguinal hernia.

This case evidently represents a good example of pure abdominal torsion of the primary type.

CONGENITAL CYSTIC DILATATION OF THE COMMON BILE DUCT

REPORT OF A CASE AND REVIEW OF THE LITERATURE *

M. M. ZINNINGER, M.D.
AND
JAMES R. CASH, M.D.
PEIPING, CHINA

Cystic enlargements of the common bile duct have been variously described as congenital, idiopathic and acquired. They have been called cysts, cystic dilatations and diverticulae of the duct. The etiology of the condition is obscure, and although many explanations have been offered, none of them seems to be completely satisfactory. Correct diagnosis before operation was apparently made only three times in the eighty-three cases recorded, and in many instances, the true condition was not recognized even at operation. Such errors will doubtless be repeated in the future, for few surgeons actually see a patient with this lesion. Therefore, unless the clinical picture is clear in the mind of the observer, it is unlikely that a correct preoperative diagnosis will be made. In contrast with this frequent failure in diagnosis, the methods of treatment have steadily improved, so that in the more recent cases, even when surgeons were not familiar with the results obtained in the past by different methods, successful procedures have been carried out with increasing frequency.

In the case to be described, certain features were found which indicated that the condition was of congenital origin. This report, therefore, may be of value in helping to determine the etiology.

REPORT OF CASE

History.—F. L. C., a Chinese woman, aged 40, was admitted to the medical service of the Peiping Union Medical College Hospital on Sept. 25, 1929. The principal complaints were pain in the epigastric region and vomiting. According to the history, she had been entirely well until four or five years previously, when she first started to have pain in the epigastrium. This, she said, was intermittent, dull and aching, and did not radiate. It usually came on from thirty minutes to an hour after eating, and was often relieved by vomiting. So far as she knew, there had never been fever or jaundice. She had found that pressure on the epigastrium, bending the body and needling the abdomen (a common practice in China) tended to reduce the severity of the pain.

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* From the Department of Surgery and the Department of Pathology, Peiping Union Medical College.

About six weeks before admission, the symptoms became greatly exaggerated. The pain was almost continuous, and at times it was so severe that the patient was said to have lost consciousness on several occasions. The vomiting also became almost continuous. Fever and chills were frequent. There was no jaundice. The stools were of normal color and consistency.

Examination.—On examination, it was noted that the patient was somewhat emaciated and looked quite sick. She was apparently having considerable pain in the abdomen. Her lips were pale and dry, and the tongue was coated. The heart and lungs were essentially normal; the blood pressure was 92 systolic and 44 diastolic. The temperature was 38 C. (100.4 F.); the pulse rate, 120, and the respiratory rate, 24. The lower half and the right upper quadrant of the abdomen showed fulness. The swelling in the lower portion of the abdomen was due to an enlarged uterus, the patient being in the fourth month of pregnancy. The abdominal wall moved normally during respiration. In the right upper portion of the abdomen, there were muscle spasm and tenderness on palpation, but a definite mass could not be felt or outlined by percussion. The liver and spleen were not felt. The upper margin of the uterus could be palpated midway between the symphysis and the umbilicus.

Laboratory examinations showed the urine and stools to be normal. Examination of the blood showed: red cells, 4,100,000; hemoglobin, 93 per cent, and white cells, 5,250, with polymorphonuclear cells, 60 per cent; lymphocytes, 36 per cent, and large mononuclear cells, 4 per cent. The icteric index was 5 units, and the van den Bergh test showed only a trace of bilirubin.

The tentative diagnosis was subacute perforation of a gastric or duodenal ulcer or disease of the gallbladder. A roentgenologic examination of the stomach was requested. This examination showed that the pyloric end of the stomach was irregular; the duodenal bulb was small, contracted and not well filled. The first portion of the duodenum was narrowed; the second portion was dilated, and there was marked duodenal stasis. There were hypotonicity and decreased motility of the stomach. Sodium tetra-iodo-phenolphthalein was given by mouth, but a shadow was not seen in the region of the gallbladder either thirteen or eighteen hours after ingestion. The roentgenologic observations were suggestive of extensive adhesions in the region of the pylorus and duodenum, probably due to an inflammatory lesion of the gallbladder.

The patient was transferred to the surgical service on September 27. On the previous night she had had a chill, after which the temperature was 40 C. (104 F.). The white blood cell count made at that time showed only 2,800 cells. On September 27, there was marked tenderness with muscle spasm in the right hypochondrium, and an indefinite, tender mass was felt about 3 cm. below the costal margin. This was thought to be either a tender, enlarged liver or a distended gallbladder. There was no jaundice. A diagnosis of acute cholecystitis with hydrops of the gallbladder was made. The patient was operated on on the following morning.

Operation.—A right rectus incision was made, and the peritoneal cavity was found free from fluid. The stomach was displaced downward and to the left by a large reddened mass which lay between the lower margin of the liver and the stomach. Exploration of the pelvis showed the uterus to be enlarged and soft (the patient was four months pregnant). No abnormalities were felt. The liver was practically normal in size, and was essentially normal in appearance and consistency except for a few whitish scars on its surface. Of great interest was a large, reddened, more or less spherical mass which lay between the liver and the stomach and duodenum. There were no adhesions surrounding it, but it was

quite red, and there was edema of its walls. The duodenum curved downward somewhat anterior to the mass; exploration showed that it lay anterior to a patent foramen of Winslow. The mass was evidently cystic. The gallbladder was slightly enlarged. Its wall was thickened and somewhat edematous, and it soon became apparent that the bladder communicated directly with the cystic mass through a large cystic duct. Palpation indicated, however, that although the outside diameter of the cystic duct was large its lumen was probably rather narrow. It seemed fairly obvious that we were dealing with an infected cyst of the common bile duct. The hepatic ducts could not be made out, the rounded mass coming off almost directly from the under surface of the liver. The head of the pancreas could be felt below the cyst and was apparently of normal consistency. Stones could not be felt within the gallbladder or within the cyst. Pads were placed to wall off the abdominal cavity, and a purse string suture was laid in the anterior wall of the cyst. A needle was inserted, and 450 cc. of slightly turbid bile was aspirated. As this was done, the cyst and the gallbladder collapsed. The cyst was then incised. The wall was about 7 mm. thick and was very vascular. It seemed to consist of two layers, an outer layer of loose areolar connective tissue richly supplied with blood vessels and a more dense inner layer. The interior of the cyst showed a few superficial ulcerations. It could not be determined grossly whether or not it was lined with mucosa. An effort was made to find the opening from the cyst into the duodenum, but this could not be discovered.

Digital exploration within the cyst failed to reveal the presence of stones. Although the patient had not had jaundice and the cyst was presumably draining into the duodenum, in the presence of the infection, it was thought advisable to secure more adequate drainage, but not to drain the bile to the exterior. The lower margin of the cyst was then dissected from the duodenum over a small area, and a lateral anastomosis was made between it and the duodenum, the stroma being about 1 cm. long. The suturing was done with a continuous lock stitch of no. 1 chromic catgut, reinforced anteriorly by mattress sutures of silk. When the duodenum was opened, there was bile-tinged fluid within its lumen. A finger was inserted through the opening in the duodenum, but even on bimanual palpation from the inside of the duodenum and the inside of the cyst, the papilla of Vater could not be located. Stones were not felt. An effort was then made to find the hepatic ducts. Viewed from the inside of the cyst, two ducts from the liver could be seen opening directly into it. These ducts were not dilated. In order to reduce the size of the cyst and also to secure tissue for microscopic examination, an elliptical area was excised from the anterior wall of the cyst. This opening was closed with a continuous lock stitch of no. 1 chromic catgut reinforced with a continuous right-angled suture of medium silk. One cigaret drain was placed down to the vicinity of the suture line, and the abdomen was closed tightly with interrupted silver wire sutures.

Course.—The patient stood the operation well. Her pain was almost entirely relieved, although she complained of discomfort at the site of the incision. She presented evidence of acidosis. There was an odor of acetone to the breath, and the urine contained acetone and diacetic acid. Blood taken at this time showed a carbon dioxide-combining power of 41.2 per cent by volume. She was given a solution of dextrose intravenously, subcutaneously, by mouth and by rectum, and her condition improved greatly.

From the bile obtained at operation, a pure culture of Bacillus paratyphosus A was grown. A Widal test made on September 30 showed agglutination to a dilution of 1: 160 with this organism. Repeated blood cultures produced no organisms.

The abdomen remained soft; the drain was removed in forty-eight hours, and the wound healed per primam intentionem. The temperature fluctuated considerably, but showed a tendency to return to normal. The white blood cell count remained low. The spleen could not be palpated. The patient continued to look very sick, although she said that she felt well.

On October 5, vomiting began and persisted in spite of all attempts at control. Acidosis again developed and could not be controlled. The patient went into a state of collapse; she died on October 9, eleven days after operation. On October 8, a positive culture of B. paratyphosus A was obtained from the stools. On the same day, the Widal test showed agglutination with this organism to a dilution of 1 : 1,280.

Autopsy.—The outstanding observations at autopsy, which was performed fourteen hours after death, were as follows:

Gross Description: The body was that of a greatly emaciated Chinese woman of middle age. In the right rectus region was a long, recently made surgical incision tightly closed by wire sutures. When this incision, through which it was necessary to perform the autopsy, was opened, the underlying abdominal viscera were found to be bound to the anterior abdominal wall by a layer of fibrin within the meshes of which was considerable purulent exudate. There was a large cystic mass about 10 cm. in diameter lying in apposition to the right inferior surface of the liver, to which the pyloric end of the stomach, the duodenum, pancreas, transverse colon and omentum and several loops of ileum were closely bound by a massive amount of fibrinopurulent exudate. The gallbladder was not seen. In the dome of the cystic mass, which was adherent to the abdominal wall, was a slightly elliptical opening, 4 cm. in diameter with slightly everted edges, from which hung a number of black silk sutures. The omentum was closely plastered about the edges of this mass of adherent structures. The remaining peritoneal surfaces were smooth and glistening. The abdominal cavity contained no free fluid. Within the uterus, which extended to the umbilicus, a fetus was easily palpated. The spleen was small and firm.

The liver was of about normal size, rather soft and a mottled, grayish brown. The capsule of the left lobe and the anterior surface of the right lobe were smooth and glistening, but the inferior surface of the right lobe, which was densely adherent to the large cystic mass already mentioned, was covered by a thick layer of fibrinopurulent exudate. On the superior surface of the liver, a number of dark red, sharply outlined zones, each about 1 cm. in diameter, which appeared to be hemorrhages, were seen within the underlying tissue. On section, the cut surface was seen to be a mottled, grayish brown; the lobules were swollen and rather indistinct in outline. Throughout the liver were a number of small, dark red, hemorrhagic zones, each embracing several lobules. In addition, countless opaque, yellowish-gray points, each less than 1 mm. in diameter, were seen, all of which seemed to lie in portal areas. Occasionally such a structure somewhat larger than the rest was seen which had a soft, purulent, bile-stained center.

In studying the mass of structures matted together with fibrin at the hilus of the liver, the duodenum was first opened. Its mucosa was deeply stained with bile and was intact except for a small opening in its posterior wall 1 cm. in diameter by which it communicated freely with the adjacent cyst. The margins of this opening were apparently completely covered by mucosa, and a number of black silk sutures were found binding the duodenum to the wall of the cyst in this region. The stomach and transverse colon were next opened and were found to be of normal appearance. The neck and proximal segment of the gallbladder, measuring 2.5 cm. in length and 8 mm. in diameter, were found to lie embedded in fibrin

closely adherent to the superior and anterior surface of the cyst. The fundus and distal segment, measuring 4 cm. in length and 1.5 cm. in diameter, after making a sharp bend posteriorly, lay collapsed between the wall of the cyst and the liver. When the gallbladder was opened, the proximal segment was found to contain about 1 cc. of thin green bile. The wall was moderately thickened, but the mucosa of the entire structure was intact and of normal appearance. By an opening 5 mm. in diameter, the gallbladder passed directly into a somewhat tortuous, tubular structure 1.5 cm. in length and from 3 to 4 mm. in diameter. This structure seemed to correspond to the cystic duct, but had an unusually thick wall (from 1 to 2 mm.) and was lined by mucosa similar to that seen in the gallbladder. No valvelike folds were present in its mucosa. This unusual cystic duct led directly, by an opening 1 cm. in diameter, into a large cystic cavity averaging about 8 cm. in diameter, the tough, stiff wall of which varied from 1 to 5 mm. in thickness. The cyst was partially divided from above and posteriorly by a low septum, 2.5 cm. in length and 2 cm. in height, into two compartments. The cystic duct opened into the right compartment, and in the posterior wall of the left compartment, separated by a distance of only a few millimeters, were found the elliptical openings of two large hepatic bile ducts. The interior aspect of the inferior portion of the cyst presented a communication, 1 cm. in diameter, with the duodenum, the result of surgical intervention. To the right of this opening was a conical pouch leading in the direction of the head of the pancreas. When this pouch was explored, a small opening about 1 mm. in diameter, partially guarded by a small, thin fold of soft tissue projecting from the wall of the cyst, was seen. A probe could not be passed into this orifice, but when it was opened carefully with scissors, a duct 3.5 cm. in length, with a thin wall and a lumen 1.3 mm. in diameter, was found passing slightly diagonally to the left through the pancreas, and opening into the duodenum by an elliptical orifice 2 mm. in length. At a point 7 mm. distant from the wall of the cyst, this duct was joined by the pancreatic duct. There seemed to be no doubt that the small structure connecting the cyst with the duodenum was at least a portion of an anomalous common bile duct. It was of approximately the same diameter throughout its course and appeared to be covered by mucosa. Its wall was very thin and delicate (fig. 1).

The cyst contained only about 3 cc. of thin, slightly turbid bile. Its lining was smooth and bile-stained throughout, having a rather velvety appearance, as if it were covered by mucosa, but showing a number of irregular shallow ulcers.

The inferior surface of the right lobe of the liver showed a marked degree of atrophy as the result of pressure by the cyst. When the larger branches of the hepatic bile ducts were dissected from their two openings into the cyst far back into the liver substance, they were found to be unaltered. No macroscopic changes were noted in the smaller bile ducts. Although the portal vein appeared to have been somewhat compressed by the posterior aspect of the cyst, no further changes were noted in it, nor was there appreciable dilatation of the splenic and mesenteric veins.

Aside from a few loops of ileum and a portion of colon adherent to the cyst at the hilus of the liver, the peritoneal surface of the intestine was smooth and glistening. A few of Peyer's patches in the lowermost portion of the ileum were slightly swollen and showed occasional shallow ulcerations. Otherwise, the mucosa was not remarkable. No parasites were present. The mesenteric lymph glands were unaltered.

The spleen weighed 80 Gm. It was quite small, dark red and rather firm. On section, the malpighian bodies were inconspicuous, the connective tissue was somewhat increased, and the rather dry pulp was scraped away with difficulty.

There was a moderate degree of brown atrophy of the heart. The aorta showed early arteriosclerosis.

The lungs were voluminous and dark red. On section, they were moist and engorged with blood.

The pancreas and suprarenal glands were not remarkable.

The kidneys were of normal size and consistency. There were a few small scars on the surface; otherwise, they were not altered.

At the request of the patient's family, the uterus, ovaries, fallopian tubes and brain were not removed. The uterus extended to the level of the umbilicus, and a fetus within it was readily palpated. The ovaries and fallopian tubes were of normal appearance. The corpus luteum of pregnancy was found in the right ovary.

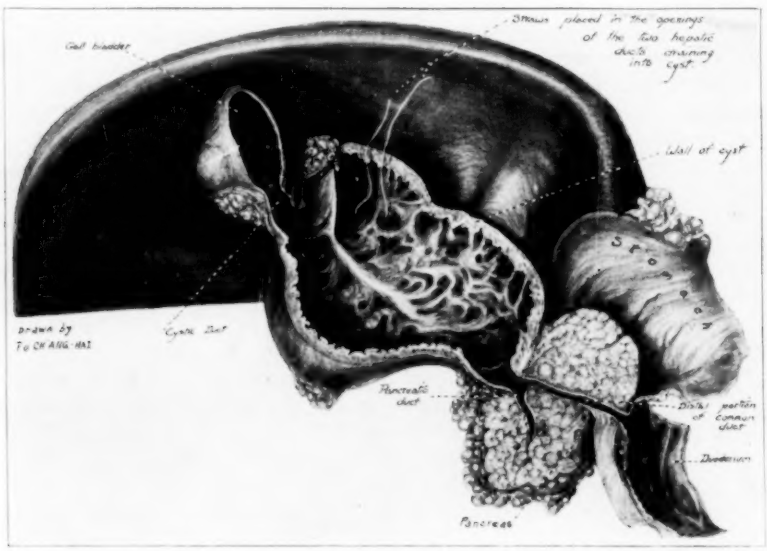

Fig. 1.—Sketch of specimen taken at necropsy, showing the relation of the cyst to the gallbladder, the hepatic ducts and the terminal portion of the common bile duct.

Microscopic Examination: The gallbladder was lined by mucosa composed of long, irregularly shaped, characteristic villi covered by a single layer of columnar epithelium. The mucosa was infiltrated with a few polymorphonuclear leukocytes and a moderate number of small mononuclear cells. The mucosa lay on a thick, loose, musculo-elastic layer which was in turn covered by a thin adventitia of connective tissue. Both the musculo-elastic layer and the adventitia contained many arteries, veins and nerves and were infiltrated slightly by lymphocytes and other small mononuclear cells. Glands were not seen in the wall of the gallbladder.

Sections of the cystic duct showed the same general characteristics as those of the gallbladder. It was found to be lined completely with mucosa, the villi of which were somewhat more slender and less irregular than those of the gallbladder, but were also covered with low cylindric epithelium. Infiltration by polymorphonuclear leukocytes and mononuclear cells was somewhat greater than

in the gallbladder. The musculo-elastic and adventitial layers were directly continuous with those of the gallbladder and were of the same character, but both layers showed considerable increase in thickness due to diffuse growth of connective tissue, and the cellular infiltration was somewhat more intense. The bundles of smooth muscle were just as large and as numerous as in the wall of the gallbladder, but were somewhat more widely separated by connective tissue and infiltrating cells.

The best sections of the wall of the cyst were made from a large, freshly fixed portion removed for study at the operation. The piece of tissue measured 8 by 5 by 0.8 cm. and was removed from the site of the opening in the dome of the cyst already described. Although it could not be stated exactly which part

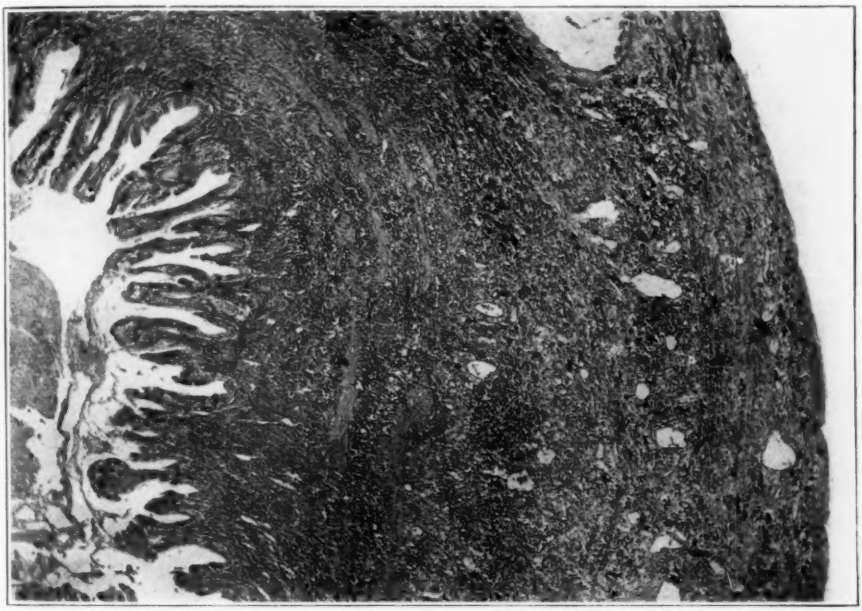

Fig. 2.—Section of a portion of the anterior wall of the cyst removed at operation, showing a well preserved mucosa and a great thickening of the wall. The concentric homogeneous layers of tissue consist of smooth muscle fibers. The remainder of the wall is composed of connective tissue and infiltrating cells; × 30.

of the wall of the distended cyst this tissue represented, it was clear from the contracted specimen that it was at least 3 cm. distant from the opening of the cystic duct.

One of the sections from this piece of tissue showed the cyst in this area to be completely lined with mucosa in all respects similar to that of the gallbladder and the cystic duct (fig. 2). The tips of many of the villi were necrotic and infiltrated with leukocytes, and overlying large areas of the mucosa was a thick layer of fibrin, leukocytes and débris. A few short, gram-negative bacilli in small clumps and also occasional gram-positive, slightly oval bacteria occurring mainly in pairs and short chains were seen within this exudate. Bacteria were not seen

within the underlying tissues, though the mucosa was densely infiltrated with leukocytes and wandering cells. Beneath the mucosa was a thick layer composed of large bundles of smooth muscle interlacing with elastic fibers and rather widely separated by a loose meshwork of fibrous tissue. Infiltration by lymphocytes and larger mononuclear cells was intense. Beyond the musculo-elastic layer was a very thick layer of dense fibrous tissue composing more than half the total thickness of the wall of the cyst. This layer contained many greatly dilated blood vessels and was also heavily infiltrated with mononuclear cells. A number of large phagocytes filled with granules of hemosiderin were also present. On the surface of the adventitia were a few strands of freshly formed fibrin.

Fig. 3.—Inner layer of the wall of the cyst from an area not showing mucosa. Marked chronic and acute inflammatory changes are present. Many large bundles of smooth muscle fibers embedded in dense fibrous tissue compose the greater part of the wall of the cyst; × 100.

Eight other sections taken from all portions of the wall of the cyst showed essentially the same structure as that just described for the specimen removed at operation, the only differences being in the condition of the mucosa. Small villi somewhat less numerous than in the gallbladder and the cystic duct were found in all of the sections. Many of these were still covered with epithelium, which was generally of the low cuboidal variety. In many of the sections most of the epithelium was missing, but in all of them except one villi at least partially covered with epithelium were present. In this one section, taken from the inferior portion of the cyst, the lining consisted of a thick layer of granulation tissue showing

marked chronic inflammatory changes and covered by fibrinopurulent exudate (fig. 3). The structure of the musculo-elastic layer was retained in all of the sections. Through one long interval, including the entire gallbladder, cystic duct and entire anterior aspect of the cyst, the musculo-elastic layer was found to be in continuity. The varying thickness of the wall of the cyst was caused mainly by variations in thickness of the outer layer of connective tissue and the degree of cellular infiltration.

Throughout the sections of liver were striking small focal necroses situated in the portal areas and frequently adjacent to the smaller bile ducts. These lesions consisted of densely coagulated central areas composed of necrotic liver cells,

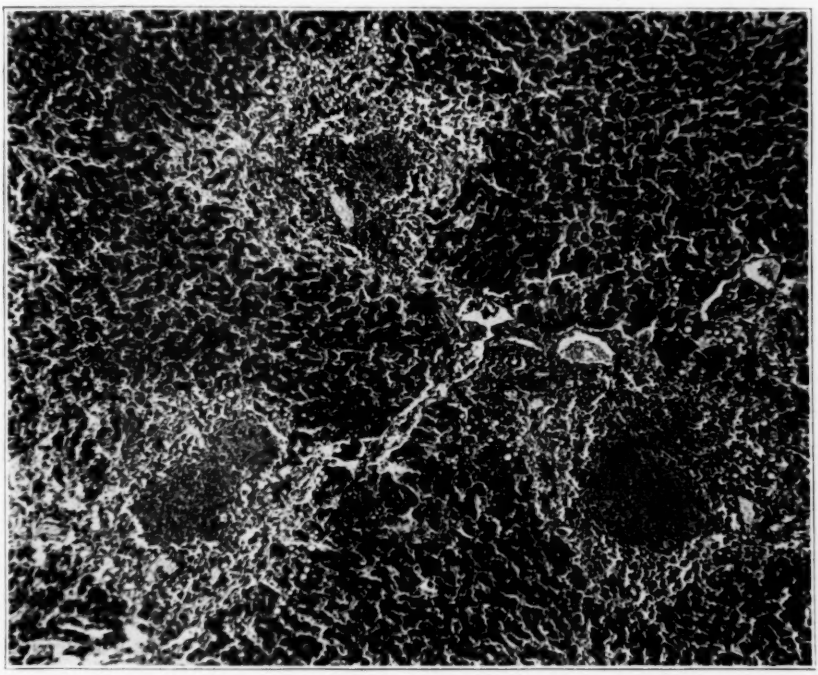

Fig. 4.—Focal necroses in the liver. Such lesions were found in each portal area; ×70.

infiltrating leukocytes and mononuclear cells surrounded by a less dense zone containing partly necrotic, shrunken and fat-laden liver cells and a few leukocytes and wandering cells. Within this loose tissue were frequent hemorrhages. Several small abscesses within which were seen bile ducts and free bile pigment were also present. A few gram-negative bacilli were found in these abscesses. There were also a few small, fresh hemorrhages which appeared to arise in the portal areas. There was moderate diffuse infiltration with mononuclear cells and a few polymorphonuclear leukocytes about all of the smaller bile ducts, but none of them showed evidence of obstruction to the flow of bile. Many of the ducts, however, contained large clumps of small gram-negative bacilli.

There was slight hyperplasia of Peyer's patches, which showed superficial ulceration of the surfaces.

The mesenteric lymph nodes were unaltered.

The colon was normal.

The lungs showed a few patches of early pneumonia.

The stroma of the spleen was moderately increased. The venules were distended with blood.

The kidneys, heart, pancreas and suprarenal glands did not show changes of importance.

Comment.—The principal question of interest in this case is the nature of the cyst that comprised such a large portion of the extrahepatic biliary tract. It is clear that this structure replaced the greater part of the common bile duct, the entire hepatic duct and perhaps a portion of the cystic duct. There were no alterations in the intrahepatic ducts, which formed two main branches at the hilus of the liver and opened by separate orifices, without obstruction, into the cyst. From the inferior portion of the cyst a very narrow duct, the wall of which consisted only of a thin layer of mucosa and a few strands of connective tissue, led directly through the head of the pancreas into the duodenum. This narrow channel, evidently representing an anomalous common bile duct, was the only exit leading from the cavity of the cyst. An elongated structure representing the gallbladder and cystic duct was found with its fundus and the distal half of its body lying firmly compressed between the wall of the cyst and the under surface of the liver. The proximal portion of the gallbladder, evidently having been pulled forward and upward by the enlarging cyst, lay on the superior aspect of this structure, with which it connected by a wide opening.

It is impossible to say just how closely any of these structures correspond to the normal structures that they appear to represent, but it at any rate seems clear that the changes described were brought about by the operation of two factors, namely, congenital malformation and subsequent infection with bacteria.

The short, narrow common bile duct, which seemed to retain its original structure, was the only portion of the primary malformation that remained unaltered. While it seems a certainty that additional anomalies must have occurred in the development of the other portions of the extrahepatic biliary tract, the subsequent changes in these structures have been so great that an opinion as to the extent and exact nature of the original malformation does not seem justified. One feels safe in assuming, however, that the congenital stenosis of the intrapancreatic portion of the common bile duct offered a constant obstruction to the flow of bile and thereby played at least a part in causing the distention of the proximal portions of the biliary tract, regardless of any congenital malformation that these structures may have suffered.

In view of the absence of jaundice in this case, one at first hesitates to consider the obstruction offered by the narrow common bile duct an

important factor in the production of the cyst. On the other hand, the lumen of the duct was so exceedingly narrow that an explanation of the failure of jaundice to appear is demanded before one can speculate further on the principles involved in the formation of the other changes. This explanation is apparently afforded by the large bands of smooth muscle found in all parts of the wall of the cyst, the contractions of which, it would seem, were adequate to force bile through the narrow common duct into the duodenum, thereby preventing the development of jaundice and at the same time temporarily relieving the pressure within the cyst. Thus it appears that obstruction to the flow of bile from the liver was prevented, though the constantly recurring distention of the extrahepatic biliary tract was followed by cystic dilatation of these structures. As suggested by the position of the gallbladder, a large portion of which was found to lie in approximately the normal position of this structure but compressed between the cyst and the liver, the size of the cyst must have increased greatly since the occurrence of the original malformation. It is hardly possible to account for any appreciable enlargement of the cyst on grounds other than obstruction to the flow of bile.

The other alterations shown by the cyst are clearly the result of long-standing bacterial infection of its contents. Proof of this is afforded by the enormous thickening of the wall of the cyst and by the growth of large amounts of connective tissue and heavy infiltration with mononuclear cells throughout all of its layers. Such an imperfect mechanism for discharging the bile as that which existed in this case was doubtless rendered still more inefficient by the added obstruction to the outflow from the cyst caused by inflammatory changes in the congenitally altered common bile duct, as well as by the increased viscosity of the infected, stagnant bile. Furthermore, it would seem that the subsequent gradual stiffening of the wall of the cyst by ever increasing connective tissue had rendered the discharge of its contents more and more difficult. An apparent desperate attempt on the part of the body to meet these difficulties is shown by the enormous hypertrophy of smooth muscle seen in sections taken from all portions of the wall of the cyst.

An added point of interest is the infection of the contents of the cyst with B. paratyphosus A, which seems to have been responsible for the illness of the patient. Though the widely spread ulceration of the mucosal lining of the cyst and other acute inflammatory changes seem clearly to have been caused by this organism, it is not unlikely that other organisms in the past played a part in the production of the more chronic changes. Whether the acute paratyphoid infection was only recently acquired or was an exacerbation of chronic infection of the cyst with this organism cannot be stated with certainty. The failure to recover

paratyphoid bacilli from the blood during the patient's entire stay in the hospital and the absence of any changes in the spleen are points that speak against a general paratyphoid infection. It seems more likely that a chronic infection of the cyst, favored by the ever increasing stagnation of bile, became active and spread widely through the liver by way of the bile passages. The low leukocyte count, as well as the rapidly increasing agglutination titer of the patient's serum for B. paratyphosus A, would be explained satisfactorily in this way.

The case is unique in that the wall of the cyst contained a far greater amount of muscle tissue and the mucosa lining the cyst was much better developed than was found to be true in other similar cases hitherto reported.

REVIEW OF THE LITERATURE

By a careful search of the literature it has been possible to find reports or references to reports of 109 cases of cystic dilatation of the extrahepatic bile ducts. No account has been taken of cylindric enlargements such as not infrequently occur after partial or complete occlusion of the common duct by stone, tumor, traumatic stricture or pancreatitis. In a few such cases there has been cystic dilatation (Fowler,[1] Eve, Wilks-Moxon, Söderlund[2] and others), but these have been excluded by most previous authors, and we have not included them. The cystic dilatations associated with complete atresia of the common duct in infants have also been excluded (Witzel, Elperin, Oxley,[3] Ylppö, Bohm, Buzik, Wickham-Legge, Parker and von der Werth), as have the dilatations of the hepatic and cystic ducts (Derwissieu, Rosenburg, Elisher, Oglobin,[4] and others), although some or all of these have been included in some reviews. We think the case reported by Flechtenmacher[5] should be excluded as one of an unproved cyst of the choledochus. In a number of reviews the case credited to Schilbe is mentioned. The only reference given is the citation by Waller, and since Waller gives no bibliographic reference it seems desirable to exclude this case. On account of its similarity to Letulle's case,[6] we are of the opinion that it is his case

1. Fowler, R. S.: Choledochus Cyst, Ann. Surg. **64**:546, 1916.

2. Söderlund: A Case of Stone in the Bile Ducts, Acta chir. Scandinav. **59**: 253, 1925.

3. Oxley, M. G. B.: Congenital Atresia of the Duodenal Opening of the Common Bile-Duct in an Infant, Producing a Large Abdominal Tumor, Lancet **2**:988 (Dec. 8) 1883.

4. Oglobin, A. A.: Idiopathic Cysts of the Bile Ducts, Vestnik khir. **9**:177, 1927; abstr., Internat. Abstract Surg., October, 1928, p. 324.

5. Flechtenmacher, C.: Operativ entfernte Choledochuszyste im Ligamentum hepato-duodenale, Wien. klin. Wchnschr. **32**:365, 1919.

6. Letulle, Maurice: Dilatation kystique des voies biliares cholangiectasies congenitales, Presse méd. **21**:97 (Feb. 1) 1913.

under the wrong name. In several reviews, the case of Bakes,[7] subequently reported by Sternberg,[8] and that of Hildebrand,[9] later reported by Kremer,[10] have been included once under each author, thus making 4 reports of 2 cases. On the other hand, we (along with others) are including the cases of Heid,[11] Heiliger,[12] Broca[13] Yamanouchi[14] and Wettwer,[15] all of which were excluded by Sénèque and Tailhefer.[16] The best reviews have been those of Waller,[17] Sénèque and Tailhefer[16] and, particularly, McWhorter.[18] A well studied case is that reported by Feyrter.[19] Of the 82 cases that we have accepted as ones of congenital cystic dilatation of the common duct, the original reports have been read in 52, the remaining cases being taken from the reviews of previous authors, principally from the papers by Waller,[17] Dreesman,[20] McWhorter,[18] Neugebauer[21] and Sénèque and Tailhefer.[16] From the last named paper were obtained 7 reports from the Polish literature (Ejalsberg, Kulakoff, Tothfalussy, Jedlicka, Ewoyan, Winternitz and Sebek) not previously reported in English. In addition to these

7. Bakes: Wien. klin. Wchnschr. **20**:298, 1907.

8. Sternberg, Carl: Cholezystostomie, Wien. klin. Wchnschr. **24**:1616, 1911.

9. Hildebrand: Geheilte Choledochuszyste, Deutsche med. Wchnschr. **39**:2224 (Nov. 6) 1913.

10. Kremer, John: Ein Fall einer durch Cholodocho-Duodenostomie dauernd geheilten echten Choledochuszyste, Arch. f. klin. Chir. **113**:99, 1919.

11. Heid: Inaugural Dissertation; cited by McWhorter, G. L.: Congenital Cystic Dilatation of the Common Bile Duct, Arch. Surg. **8**:604 (March) 1924.

12. Heiliger: Inaugural Dissertation; cited by McWhorter, G. L.: Congenital Cystic Dilatation of the Common Bile Duct, Arch. Surg. **8**:604 (March) 1924.

13. Broca: Bull. et mém. Soc. nat. de chir. **23**:209, 1897.

14. Yamanouchi, K.: Ueber die cystische Dilatation des Ductus choledochus, Ztschr. f. Jap. Chir. Gesellsch., 1921, vol. 22; abstr., Zentralorg. f. d. ges. Chir. **16**:228, 1922; cited by Neugebauer, F.: Beitr. z. klin. Chir. **131**:448, 1924; cited by Judd, E. S., and Greene, E. J.: Surg., Gynec. & Obst. **46**:317, 1928; cited by Willis, B. C.: Ann. Surg. **87**:48, 1928.

15. Wettwer: Ein Fall von kongenitalen Choledochuscyste, Dissertation, Göttingen, 1907; cited by Waller, E.: Ann. Surg. **66**:446, 1917.

16. Sénèque, J., and Tailhefer, A.: Les dilatations congénitales du cholédoque, J. de chir. **33**:154, 1929.

17. Waller, Erik: Idiopathic Choledochus Cyst, Ann. Surg. **66**:446, 1917.

18. McWhorter, G. L.: Congenital Cystic Dilatation of the Common Bile Duct, Arch. Surg. **8**:604 (March) 1924.

19. Feyrter, F.: Ueber Fehlbildungen der extrahepatalen Gallenwege mit Störung der normalen Hohlraumbildung (Verödung, Enge und cystische Erweiterung) und des normalen Gangverlaufes, Virchows Arch. f. path. Anat. **271**:20, 1929.

20. Dreesman: Beitrag zur Kenntnis der kongenitalen Anomalien der Gallenwege, Deutsche Ztschr. f. Chir. **92**:401, 1908.

21. Neugebauer, F.: Zur Kenntnis der idiopathischen Choledochuscyste, Beitr. z. klin. Chir. **131**:448, 1924.

82, references to 6 other reports were obtained, all of which probably should be included, but neither the original reports nor satisfactory reviews were available (di Natale,[22] Jauregui,[23] Olivecrona,[24] Sieber,[25] Stoney[26] and Zaczek[27]). Including our own case, therefore, there are 83 cases available for study.

Symptomatology.—The disease occurs predominantly in females. Sixty-eight of the 83 patients were females and 14 males and in 1 case the sex was not recorded. It is a disease of children and young adults. The ages of the 69 patients when they presented themselves for treatment were as follows: from birth to 10 years, 28 cases; from 11 to 20 years, 22 cases; from 21 to 30 years, 20 cases; from 31 to 40 years, 7 cases; over 40 years, 4 cases. The oldest patient was 56, but she knew that she had had a tumor of the abdomen for thirty-six years. In many instances, jaundice, abdominal pain or tumor was present in infancy or in childhood for a short time, and then disappeared, only to recur later and cause the patient to apply for treatment. In other cases, the attacks were intermittent over a period of many years.

The three most prominent symptoms have been tumor, jaundice and pain. An abdominal tumor was present in 60 of the 83 cases, indefinite in 5, absent or not felt in 4 and not reported in 14. Jaundice was present in 60 of the patients at the time they came under observation, and had been present in 2 others previously. It was absent and had never been noticed in 7, and was not reported in 14. In 8 it was noted as slight or very slight. There was a variation from complete absence to the intense jaundice of long-standing, complete obstruction. In many cases it was intermittent or noted on more than one occasion. Pain was reported as present in 43 cases, absent in 5 and not noted in 35. It was slight in some cases, but more often was sharp and colic-like. Jaundice and pain were associated in many instances, the jaundice appearing shortly after the pain. The stools were acholic in only a small proportion of cases, in which there was deep jaundice. In 7 cases the symptoms were accentuated by pregnancy or they appeared during or just after the beginning of pregnancy (Seyffert,[28] Dreesman,[20]

22. di Natale, L.: Cisti del coledoco, Arch. ital. di chir. **23**:553 (May) 1929; abstr., Brit. M. J. **2**:30 (Aug. 24) 1929.

23. Jauregui, P.: Cyst of the Common Bile Duct, Rev. de cir. **7**:536, 1928.

24. Olivecrona, H.: Infected Cyst of the Common Bile Duct, Operation, Recovery, Acta chir. Scandinav. **62**:347, 1927.

25. Sieber, F.: Large Cyst of the Bile Ducts, Zentralbl. f. Gynäk. **53**:2848, 1929.

26. Stoney, R. A.: Cyst of the Common Bile-Duct, Irish J. M. Sc., December, 1929, p. 764.

27. Zaczek: Congenital Cyst of the Common Bile-Duct, Polski Przegl. Chir. **6**:639, 1927.

28. Seyffert, cited by McWhorter (footnote 18).

Goldammer,[29] Hildebrand,[9] McWhorter,[18] Bachkiroff[30] and Zinninger and Cash). In the case reported by Sénèque and Tailhefer, the symptoms were relieved by pregnancy.

Fever was fairly frequent but not constant. It was mild in the majority of cases, and severe in only a few when definite infection of the contents of the cyst was present.

Diagnosis.—The preoperative diagnosis has usually been rather obscure, and in many instances either no definite diagnosis was made or else several were suggested. The correct diagnosis has been made before operation in only 3 cases (Iselin,[31] Neugebauer[21] and Taylor[32]). In a fourth case, the correct diagnosis was considered, only to be rejected in favor of that of a more common condition (Wright[33]). The most frequent diagnosis made has been echinococcus cyst (eighteen times). Other diagnoses in the order of frequency have been: pancreatic cyst, seven times; stone in the common duct, six times; mesenteric cyst, three times; cholecystitis, four times; enlarged gallbladder, three times; malignant growth, tuberculosis and cyst of the kidney, twice each, and retroperitoneal tumor, intestinal obstruction, ovarian cyst, catarrhal jaundice, perforation of the gallbladder and cyst of the liver, once each. In 35 cases, either no diagnosis was made or else it was not noted.

Even at operation the true condition was not recognized in a number of instances, and in nearly every one of these the termination was fatal. Five patients were treated by excision without anastomosis of the hepatic duct to the intestine, while in a number the tumors were drained as supposed echinococcus cysts, pancreatic cysts, cysts of the liver or distended gallbladders.

Pathology.—The gross appearance was much the same in all cases, and one is struck by the similarity of the observations made at operation. The large, tense, cystic tumor, generally spherical, lay in the upper part of the abdomen just below the liver and pushed the stomach downward and to the left and the duodenum downward. The position was retroperitoneal, sometimes in front of the duodenum and sometimes behind it. The gallbladder was generally found to be either small or of normal size, and sometimes was not seen at operation. In all except 2

29. Goldammer: Beiträge zur Chirurgie der Gallenwege, Beitr. z. klin. Chir. **55**:214, 1907.

30. Bachkiroff: Sur la pathogénie des kystes du cholédoque, Chir. Arch. Veliaminova. **27**:63, 1911; cited by Sénèque and Tailhefer (footnote 16).

31. Iselin, Hans: Gemeinsame Cyste der Gallen-und Pankreaswege, Arch. f. klin. Chir. **145**:304, 1927.

32. Taylor, Julian: Cystic Dilatation of the Common Bile-Duct: Record of an Example, Brit. J. Surg. **16**:327, 1928.

33. Wright, H. W. S.: Congenital Diverticulum of the Common Bile Duct, Surg., Gynec. & Obst. **39**:156, 1924.

(Seeliger [34] and Hill and Ramsay [35]) of the cases in which the details were noted, the cyst involved only the extraduodenal portion of the duct, and the lower intraduodenal portion varied in length from 0.7 to 4 cm. In these 2 cases, the authors believed that the dilatation involved the entire length of the duct. In 3 other instances (Iselin,[31] Mayesima [36] and Matthieu [37]), the pancreatic duct entered the cyst, so that the dilatation probably involved at least part of the intraduodenal portion. Seeliger [34] also thought that in his case the pancreatic duct entered the cyst, but he was not quite sure that it did so.

The cysts have varied greatly in size. The smallest was in a still-born fetus born nearly at term which was described by Heiliger;[12] the cyst measured 3 by 2.5 cm. The largest was estimated to contain 8 liters (Reel and Burrell [38]). Twelve of the cysts for which the quantity of fluid was recorded held from 500 cc. to 1 liter; eleven, from 1 to 2 liters; seven, from 2 to 3 liters; three, from 3 to 4 liters, and four, more than 4 liters.

The wall of the cyst has varied considerably in thickness and in structure. In 9 cases it was found to be from 2 to 3 mm. in thickness; in 3, from 4 to 5 mm., and in 2 from 7 to 7.5 mm. In several cases it was described as thick, and in others as thin; in 4 cases it was described as very thin, and in 2 of these (Waller [17] and Reel and Burrell [38]) it was ruptured during the operation. The wall was generally vascular, and has been described grossly as made up of fibrous tissue which seemed to be lined by mucosa. However, in only 7 of the cases was any epithelial lining demonstrated by microscopic examination, and in no instance was the epithelium well developed. It is striking that in 24 cases it was definitely stated that in the histologic preparations no epithelium was found lining the inside of the cyst. The rapid distintegration of the epithelium that lines the larger biliary passages is well known, and it is not unreasonable to suppose that this has been a factor in the large number of instances in which no epithelium was found. All reports of microscopic examination state that the wall was largely composed of connective tissue. In 12 cases it was stated that muscle was not present, while in 9 more there was only the statement that the wall was made up of connective tissue, so that presumably muscle was absent in these cases as well.

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34. Seeliger, S.: Beitrag zur Kenntnis der echten Choledochuscysten, Beitr. z. klin. Chir. **99**:158, 1916.

35. Hill, N. H., and Ramsay, R. A.: Cystic Dilatation of the Common Bile Duct, Brit. M. J. **2**:991 (Nov. 28) 1925.

36. Mayesima, J.: Zur Kasuistik der primären cystischen Erweiterung des Ductus choledochus, Deutsche Ztschr. f. Chir. **119**:338, 1912.

37. Matthieu, J.: Congenital Cyst of the Common Bile Duct, Ann. Surg. **92**: 159, 1930.

38. Reel, P. J., and Burrell, N. E.: Cystic Dilatation of the Common Bile Duct, Ann. Surg. **75**:191, 1922.

At operation, the opening of the cyst into the duodenum was definitely demonstrated in only 1 case (Waller [17]). At autopsy a great variety of conditions has been found in the terminal portion of the duct and at the outlet of the cyst. In 17 cases, a valvelike fold was found over the outlet, and in a number of these there were similar folds over the mouths of the hepatic and cystic ducts. (McWhorter [18] included the cases of Ebner [39] and Waller [17] in this group, but we have been unable to confirm this.) The course of the duct through the duodenum was somewhat abnormal in a large number of cases. In 11, there were one or more sharp kinks, and in 13 there was narrowing or stenosis.

It is hard to arrive at a definite conclusion as to how frequently there was a complete atresia of the duct. In many cases, no opening was found between the cyst and the intestine at operation, but the clinical history and the preoperative findings precluded the assumption of complete obstruction, and in others in which no opening was found at operation, it was found later at autopsy. In the case reported by Butters,[40] there was deep jaundice, but the data are incomplete. In the case reported by Rolleston,[41] only the pathologic specimen was presented, without a clinical history. In the cases reported by Seeliger,[34] Wyllie [42] and Giezendanner,[43] an opening was not found at autopsy, even by serial sections in Giezendanner's case, but in none of these was there clinical evidence of complete obstruction. In the case reported by McWhorter,[18] no opening was found at operation during excision of the cyst, but again the symptoms and signs were not those of total obstruction. In the cases reported by Hildebrand [9] and Swain,[44] only the changes found at operation were given, but deep jaundice and acholic stools were present, so that a total occlusion of some sort must have existed. In the case reported by Lavenson,[45] there was deep jaundice with acholic stools for eight months, and at autopsy the duct was observed as a definite structure, the lumen of which was completely obliterated for a considerable distance. In a large number of cases an obstruction could not be demonstrated.

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39. Ebner, A.: Idiopathische Choledochuscyste und Purpura haemorrhagica fulminans, Beitr. z. klin. Chir. **64**:473, 1909.

40. Butters: Choledochus-Zysten, Deutsche med. Wchnschr. **36**:1351 (July 14) 1910.

41. Rolleston, Humphry D.: Diseases of the Liver, Gall-Bladder and Bile Ducts, ed. 3, New York, The Macmillan Company, 1905, p. 651.

42. Wyllie, W. G.: Congenital Cyst of the Common Bile-Duct, Lancet **1**:1342, 1925.

43. Giezendanner, Ernst: Beitrag zum Studium der kongenitalen Choledochuszysten, Frankfurt. Ztschr. f. Path. **38**:150, 1929.

44. Swain, W. P.: A Case of Cholecystenterostomy with the Use of Murphy's Button, Lancet **1**:743 (March 23) 1895.

45. Lavenson, R. S.: Cysts of the Common Bile Duct, Am. J. M. Sc. **137**:563, 1909.

The contents of the cyst were reported to be infected in 14 cases (Arnolds,[46] Bolle,[47] Brun and Hartman,[48] Budde,[49] Dreesman,[20] Iselin,[31] Krabbel[50] (?), Letulle,[6] Schürholz,[51] Sebek,[52] Taylor,[32] Wagner,[53] Zipf[54] and Zinninger and Cash).

The condition of the liver was not uniformly characteristic and apparently depended on the degree of obstruction. In the cases in which there was deep jaundice, biliary cirrhosis or interstitial hepatitis was described. In 4 cases, there were areas of focal necrosis in the liver (Mayesima,[36] Lange,[55] Zipf[54] and Zinninger and Cash).

Treatment.—The various types of treatment and the results are summarized in table 1.

TABLE 1.—*Types of Treatment in Cases Reported in the Literature*

Treatment	Number	Died	Recovered
No treatment	8	8	0
Aspiration	3*	3	0
Drainage only	27	24†	3‡
Drainage followed by secondary anastomosis	14	5	9
Primary anastomosis of cyst to intestine	18	5	13
Excision of cyst; primary anastomosis of hepatic duct to intestine	3	1	2
Excision of cyst with drainage; secondary anastomosis to intestine	1	0	1
Excision or attempted excision of cyst, with or without drainage	6	6	0
Nature of operation not known	3	2§	..
Total	83	54	28

* Laparotomy was performed in one case.
† Three patients died after from one to three months of hemorrhage from fistula.
‡ One patient died after three years of pulmonary tuberculosis; 1 was well four years, and 1 was well after one and one-half years.
§ The outcome of one case was not known.

46. Arnolds: Manneskopfgrossen Retentionscyste des Choledochus, Deutsche med. Wchnschr. **32**:1804 (Nov. 1) 1906.

47. Bolle, H.: Ein Fall von idiopathischer Choledochuszyste, Deutsche med. Wchnschr. **48**:1381 (Oct. 13) 1922.

48. Brun and Hartman: Bull. et mém. Soc. nat. de chir. **23**:207, 1897.

49. Budde, Max: Ueber die Pathogenese und das Krankheitsbild der cystischen Gallergangerweiterung (sogenannte idiopatische Choledochuscyste), Deutsche Ztschr. f. Chir. **157**:364, 1920.

50. Krabbel, Max: Zum operativen Heilung der idiopathischen Choledochuscyste, Beitr. z. klin. Chir. **130**:159, 1924.

51. Schürholz: Ein Fall von sogenannten idiopatischer Choledochuscyste, Arch. f. klin. Chir. **118**:91, 1921.

52. Sebek: O pricinach ysiniku, pathologii a symptomologii tak zvané idiopathické cysty choledochu, Časop. lék. česk. **66**:1720, 1927; cited by Sénèque and Tailhefer (footnote 16).

53. Wagner, A.: Beitrag zur Chirurgie der Gallenwege, Deutsche Ztschr. f. Chir. **145**:15, 1918.

54. Zipf, Karl: Ueber idiopathische Choledochuscysten, Arch. f. klin. Chir. **122**:615, 1922.

55. Lange, Kurt: Beitrag zur idiopathischen Choledochuscyste, Zentralbl. f. Chir. **54**:2287, 1927.

Aspiration alone was performed in only 3 cases, and all of the patients died. In several instances this procedure was followed by leakage of bile into the peritoneal cavity, necessitating laparotomy. In 8 cases, because of the extreme illness of the patient, no treatment for the specific condition was given. These cases also terminated fatally.

Drainage, generally by marsupialization, was the procedure most frequently followed—in 41 cases in all. In 27 of these it was the only procedure used, and of this number only 3 patients lived more than three months. Clairmont's [56] patient died three years later of pulmonary tuberculosis, with a persistent biliary fistula, but with bile in the stools. In McConnell's [57] case, the biliary fistula opened and closed several times, and a second laparotomy was done because of loss of appetite and occasional abdominal pain; however, except for the exploration, nothing was done. The patient was reported as well four years after the first operation. In the case reported by Reel and Burrell,[38] the fistula closed, and the patient was reported as well a year and a half after operation.

Fourteen of the patients in whom drainage was done had a subsequent anastomosis between the cyst and the intestine. Of these, 5 died and 9 survived, but 1 (Hildebrand [9]) had to have a third operation to enlarge the stoma made at the second operation. In nearly every instance in which death occurred following the second operation, it came shortly after operation, presumably from shock. In a number of cases in which the patients were very ill, drainage alone was carried out at the first operation with the idea of performing an anastomosis when the jaundice was less intense. However, most of the patients died too soon to permit of a second operation.

In 18 cases an anastomosis of the cyst to some portion of the intestine was done at the first operation, and in 3 others a partial or complete excision of the cyst was made, with a primary anastomosis of the stump of the hepatic duct to the intestine. In the 21 cases in which a primary anastomosis was done, there were 6 deaths and 15 recoveries.

Excision of the cyst without anastomosis was done in 6 cases, all of which terminated fatally. In 1 case excision of the cyst with drainage of the hepatic ducts was carried out, followed by anastomosis of

56. Clairmont: Zystische Dilatation des Ductus choledochus, Wien. klin. Wchnschr. **24**:1616, 1911.

57. McConnell, A.: Cyst of the Common Bile Duct, Brit. J. Surg. **7**:520 (April) 1920.

TABLE 2.—*Summary of Eighty-Three Cases Cited*

Number	Author	Year	Sex	Age	Tumor	Jaundice	Pain	Diagnosis	Treatment	Result
1	Adam, L.: Idiopathische Choledochuscyste, Zentralbl. f. Chir. **50**: 997, 1923	1923	F	31	+	+	+	Bile cyst; pancreatic cyst	1. Drainage; 2. choledochoduodenostomy 3 months later	Recovered; well 1 year later
2	Arnison, cited by Robson, A. W. Mayo: Diseases of the Gall Bladder and Bile Ducts, ed. 3, New York, William Wood & Company, 1904, p. 200; cited by McWhorter [18]	1891	?	?	+	+	?	Pancreatic cyst	Drainage	Died, 1 day after operation
3	Arnolds [46]	1906	F	13	+	+ 2 months	+	Echinococcus; a malignant condition	Drainage	Died, 1 day after operation
4	Ashby, H.: M. Chron. **10**: 28, 1898; cited by McWhorter [18]; cited by Rolleston [41]	1898	F	7	+	+ 2½ years	?	?	Aspiration ten times in 3 months; then an attempt to anastomose	Died, 1 day after operation
5	Ashby, H. T., and Platt, H., cited by Morley, J.: Brit. J. Surg. **10**: 413, 1923	1923	F	6½	?	+	+		1. Drainage; 2. anastomosis	Died
6	Bachkiroff [39]	1911	F	21	+	..	..	Echinococcus cyst of liver	Attempt to resect	Died of hemorrhage
7	Bakes [7] and Sternberg [8]	1907 and 1911	F	22	0	+	+	Pancreatic or mesenteric cyst	Primary anastomosis	Recovered; died 3 years later of other causes; autopsy by Sternberg
8	Bohmansson, C.: Cyst of the Common Bile Duct, Acta chir. Scandinav. **56**: 440, 1924	1923	M	3½	+	+	..	Retroperitoneal tumor	Primary anastomosis	Recovered
9	Bolle [47]	1922	F	13	+	Slight	++	Congestion of liver; tuberculosis; peritonitis; echinococcus	1. Aspiration; 2. drainage; 3. anastomosis; gallbladder removed	Recovered (5 weeks)
10	Broca [13]	1897	F	10	+	Slight	..	Generalized tuberculosis	For tuberculosis	Died
11	Brun and Hartman [48]	1897	F	3½	+	0	?	Infected echinococcus	1. Drainage; 2. anastomosis	Recovered
12	Budde [49]	1920	F	22	+	+	+		Drainage	Died
13	Butters [49]	1910	M	5	+	+	..	?	Drainage	Died, several days after operation
14	Clairmont [56]	1911	M	22	+	+	..	?	Drainage	Recovery; died, 3 years later of pulmonary tuberculosis
15	Douglas [65]	1852	F	17	+	+	..	?	Aspiration	Died, 14 days after operation
16	Dreesman [20]	1906 1908	F	24	+	+ 2 months	++	Echinococcus	1. Drainage; 2. anastomosis 3 months later	Died, 3 days after operation
17	Ebner [39]	1908	F	18	+	+ 6 months	++	Echinococcus or cyst of the liver	Drainage	3 months later severe purpura developed, and patient died in 4 days of hemorrhage
18	Edgeworth [63]	1895	F	4½	+	+	..	Enlarged gallbladder, with partial obstruction of common duct	Drainage of gallbladder supposedly	Died, 1 week after operation

19	Ejalsberg: cited by Sebeke [52]	1924	F	40	..	..	..	?	Excision and anastomosis	Died
20	Ewoyan [71]	1925	M	6	..	..	..	?	Excision and drainage	Died after 3 months, of hemorrhage
21	Exner, A.: Choledochuscyste, Wien. klin. Wchnschr. **24**: 1615, 1911	1911	F	23	..	+	..	Pancreatic cyst	Drainage	Died (hepatic insufficiency?)
22	Fenger: Tr. Am. S. A. **14**: 699, 1896; cited by McWhorter [18]	1896	F	33	+	+	..	Stone in common duct	Cyst opened and closed, gallbladder drained	Died, 24 hours after operation
23	Feyrter [19]	1929	F	7	..	..	..	Intestinal occlusion	Not treated	Died
24	Geizendanner [13]	1929	F	4	+ 3½ months	+ Developed slowly	+	?	No operation	Died
25	Goldammer [20]	1907	F	21	+ 3 months	+ 3 months	+	Echinococcus	Excision of cyst	Died, several hours after operation
26	Held [11]	1893	F	14	+ 6 weeks	+ 6 weeks	+ 6 weeks		No operation	Died, of diphtheria and erysipelas
27	Heiliger [12]	1910	M	Fetus	..	0	..		No operation	
28	Hildebrand [9] and Kremer [10]	1913 and 1918	F	18	+	++	..	Cyst in region of liver, with compression of common duct	1. Drainage; 2. anastomosis; 3. larger anastomosis	Recovered; presented 5 years later by Kremer
29	Hill and Ramsey [35]	1925	F	2	..	0 Had at 9 months	?	Cyst of right kidney	Drainage	Died, 22 hours after operation
30	Ipsen [70]	1913	F	18	+	+ 6 weeks	+ 6 weeks	Stone of common duct, with enlarged liver	Drainage	Died, 1 month after operation, of hemorrhage from cyst
31	Iselin [31]	1927 Patient seen in 1918	M	36	+	Repeated attacks	..	Cyst of common duct	Hepatic duct implanted into duodenum; partial excision of cyst; drainage	Recovered; pancreatic fistula reopened in 1927
32	Jedlicka, cited by Sebek [52]	1925	F	45	..	..	..	?	Primary anastomosis	Cured
33	Judd, E. S., and Greene, E. L.: Choledochus Cyst, Surg., Gynec. & Obst. **46**: 317, 1928	1928	F	13	+	Slight	+	Cholecystitis with stones	Primary anastomosis; drainage of gallbladder	Recovered
34	Kolb [62]	1905	F	10	+	+ Several months	..	Cyst, echinococcus(?)	1. Aspiration; 2. drainage	Died, 1 month after operation
35	Konitzky [59]	1888	F	21	+	+ 6 months	..	?	Drainage	Died, 8 days after operation, pulmonary embolus
36	Körte [72]	1904	F	26	..	++	+		Primary anastomosis with drainage	Died, 4 days after operation, of hemorrhage from aneurysm of hepatic artery in wall of cyst
37	Krabbel [50]	1924	F	8	+		+	Echinococcus; abscess of liver	1. Drainage; 2. anastomosis; 3. closure of fistula	Recovered

TABLE 2.—*Summary of Eighty-Three Cases Cited—Continued*

Number	Author	Year	Sex	Age	Tumor	Jaundice	Pain	Diagnosis	Treatment	Result
38	Kulakoff, cited by Sebek [52]	1924	F	21	..	..	..		Drainage	Died
39	Lange [55]	1927	F	21	+	+ 4 months	+	Pancreatic or mesenteric cyst	Drainage	Died, 2 days after operation
40	Lavenson [45]	1909	F	8	+ 3 weeks	+ 1 year	0	?	Excision	Died, 3 days after operation
41	Letulle [6]	1913	F	25	+	+	0	Echinococcus	Drainage	Died, 8 days after operation
42	McConnell [57]	1919	F	11	+	+ Very slight	+	?	1. Drainage; 2. drainage; 3. exploration, cyst size of walnut found, nothing done	Recovered; patient well and wound healed after 4 years
43	McWhorter [18]	1924	F	49	+	+ Attacks	+	Stone in common duct	Excision of cyst and gallbladder, and anastomosis of hepatic duct to duodenum	Recovered
44	Matthieu [37]	1930	M	6 weeks	+	++	?		Not treated	Died
45	Mayesima [36]	1912	M	2	+	+ 4 months	?	?	Laparotomy; cyst aspirated; no drainage	Died, 3 months after operation
46	Melichoff: Dilatation idiopathique du cholédoque, Russk. klin. **3**: 74, 1925; cited by Sénèque and Tailhefer [16]	1925	F	32	0	+	+	Cholecystitis	1. Anastomosis to stomach; 2. gastroenterostomy	Recovered
47	Morley, John: Congenital Cyst of the Common Bile Duct, Brit. J. Surg. **10**: 413, 1923	1923	F	17	+	Once	+	Ovarian cyst	1. Drainage; 2. anastomosis 3 weeks later	Recovered
48	Moynihan, (McWhorter) Guy's Hosp. Museum No. 1419	1904	F	21	..	+	2½ years	Stenosis; valve formation; cystic duct dilated; hepatic ducts dilated	Two aspirations; operation for drainage	Died, 2 days after operation
49	Neugebauer [21]	1924	F	21	+	+	+	Choledochus cyst	Primary anastomosis	Died, 5 days after operation
50	Nicolaysen, J.: Tumor cysticus ductus hepaticici et choledochi dilate, Zentralbl. f. Chir. **16**: 1269, 1899; cited by Waller [17]; cited by McWhorter [18]	1899	F	8	+	++	..		Packing against cyst; drainage 6 days later	Died, 1 day after operation
51	Pamperl, R.: Zur Behandlung der idiopathischen Choledochuszyste, Med. Klin. **21**: 1608, 1925	1925	M	23	+	+	..	None	Primary anastomosis	Recovered
52	de Quervain: Spezielle chirurgische Diagnostik, 1922, p. 398; quoted by Glezendanner [43]	1922	F	?	?	..	+		?	?
53	Reel and Burrell [38]	1922	F	56	+ 36 years	+ 4 years at times	+	?	Tube drainage	Well, 1½ years after operation

54	Roberts: Thése, Lausanne, 1914; cited by McWhorter [18]	1914	F	2	+	+	0	?	Drainage	Died, 1 week after operation
55	Rolleston [41]; Museum specimen, Newcastle-on-Tyne, no. 382-2	1905	F	9	..	..	..		No operation	Died
56	Rostowzew [58]	1902	F	13	+ 3 years	+ 3 weeks	..	Echinococcus cyst	Drainage	Died, 1 day after operation
57	Rotgans [59]	1913	F	5	0	+ intermittent	+	Cholecystitis	Drainage and cholecystectomy; anastomosis 3 months later	Recovered
58	Russell [60]	1897	M	8	+ 3 days	+ 4 days	+ 4 days	Echinococcus (catarrhal jaundice)	Drainage	Died, 4 days after operation, of hemorrhage
59	Schloessmann [61]	1911	F	7	+	+ 14 days	+ 18 days	Echinococcus cyst	Aspiration (signs of leakage); drainage	Died, 6 days after operation
60	Schürholz [51]	1921	F	3	+	+	+ 6 weeks	Echinococcus	1. Drainage; 2. anastomosis 6 weeks later	Died, 3 hours after operation
61	Sebek [52]	1927	F	18	+	+	+	Stone in common duct	1. Excision and drainage; 2. anastomosis	Recovered
62	Seeliger[34]	1916	F	13	+	+ 4 days	+ 1 month	Echinococcus; perforated gallbladder (?)	1. Drainage, delayed 3 days; 2. opening into duodenum made	Died, 6 days after operation
63	Sénèque and Tailhefer [16]	1929	F	32	+	+	+	Cholecystitis	Primary anastomosis	Recovered
64	Seyffert [28]	1888	F	23	+ 1 year	+ 2 years	+ Colic 1 year	Pancreatic cyst	Drainage	Died, 1 month after operation, of hemorrhage
65	Smit: Et tilfaelde of idiopathisk choledochuscyste, Medicinsk Rev. **32**: 285, 1915; cited by Waller [17]	1915	F	17	+ 5 years	0	+	Cyst in liver	Excision	Died, 8 days after operation
66	Swain [44]	1895	F	17	+	+ 10 months	0	Enlarged gallbladder	1. Aspiration; 2. button for intestinal anastomosis	Recovered
67	Taylor [32]	1928	F	23	+	+	+	Cystic dilatation of common duct	Anastomosis to stomach	Recovered
68	Tothfalussy, cited by Sebek [52]	1925	M	12	..	..	..		Excision and drainage	Died
69	Wagner [53]	1918	F	47	+	+	+ 6 months	Malignant condition of gallbladder or liver; stone in common duct	Button for intestinal anastomosis	Died, 4 days after operation

TABLE 2.—*Summary of Eighty-Three Cases Cited—Continued*

Number	Author	Year	Sex	Age	Tumor	Jaundice	Pain	Diagnosis	Treatment	Result
70	Waller [17]	1917	F	10	+	0	+ began at age of 3	Indeterminate	Primary anastomosis	Recovered
71	Walzel, P.: Idiopathische Choledochuscyste, Zentralbl. f. Chir. **48**: 594, 1921	1921	F	27	..	..	..	?	1. Excision of gallbladder and cyst—hepatic duct drained to surface; 2. anastomosis of hepatic duct to stomach; two subsequent operations	Hemorrhage; died
72	Weiss [64]	1909	M	6	+ 7 months	+ Slight, intermittent	+	Echinococcus	1. Diagnostic puncture; 2. exposure and suture; 3. drainage	Died, ninth day after operation
73	Wettwer [15]	1905	F	15	+	Intermittent 8 years	8 years	?	None	Died
74	Willis [66]	1928	M	12	+	0	6 months	Empyema or hydrops of gallbladder	Primary anastomosis	Recovered
75	Wilson [67]	1930	F	14	+	0	+	None reported	Primary anastomosis	Recovered
76	Winternitz, cited by Sebek [52]	1926	F	26	..	..	..		Primary anastomosis	Recovered
77	Wright [33]	1924	M	15	+	+ 5 months	Slight	Cyst of common duct (?); rejected; stone in common duct	Primary anastomosis	Died, 3 days after operation, of cholemia (?)
78	Wyllie [42]	1925	F	6	+ 1 year	+ 1 year, intermittent	?	?	Drainage; anastomosis	Died, 48 hours after operation
79	Yamanouchi [14]	1921	F	4	..	..	..	None	Operation	Died
80	Yamanouchi [14]	1921	F	16	+	..	..	None	Operation	Died
81	Zimmer, Hans: Idiopathische Choledochuscyste, Zentralbl. f. Chir. **51**: 424, 1924	1924	F	22	+ 1 month	+ Developed Gradually	+	Pancreatic cyst; echinococcus; mesenteric cyst; cyst of kidney	1. Drainage; 2. anastomosis 1 month later	Recovered
82	Zipf [54]	1922	F	14	+	Slight	+ 3 days		Drainage	Died, 3 days after operation
83	Authors' case	1930	F	40	Indefinite	0	+ 4 to 5 years; acute 1 month	Hydrops of gallbladder	Primary anastomosis	Died, 12 days after operation of acidosis

the stump of the hepatic duct to the intestine at a second operation. This patient recovered.

The total mortality in the 83 cases was 65 per cent (54 cases). In the cases in which the patients were not treated, in those in which they were treated by aspiration alone and in those in which they were treated by excision alone, the mortality was 100 per cent. In the 27 cases in which the patients were treated by drainage alone, the mortality was 88 per cent. In the 14 cases in which a secondary anastomosis was done, the mortality was 35 per cent; in the 21 in which a primary anastomosis was made, it was 28.9 per cent.

From these results one may conclude that internal drainage should be done in all cases and that anastomosis at the primary operation apparently gives better results and a lower mortality than at a second operation. This is striking if one also considers the high mortality in the cases in which the patients were treated by drainage alone, as only those who survived that operation for some time were available for secondary operations. It is unfortunate that the late end-results are known in only a few cases. The other striking feature is the fact that failure to recognize the true nature of the condition at operation has been an important factor in the high mortality. From an anatomic and physiologic point of view, it would seem that excision of the cyst with anastomosis of the hepatic duct to the intestine should be the method of choice, but unfortunately this procedure is rarely feasible for two reasons. In the first place, in deeply jaundiced patients such an operation would probably lead to fatal hemorrhage, and in the second place, the cysts usually arise directly beneath the liver, so that no extrahepatic portion of the hepatic duct is available for anastomosis.

Etiology.—The cause of cystic dilatation of the common bile duct remains obscure, though most authors think that a congenital factor is present. Various authors considered the following causes as productive of the condition:

1. Congenital malformation (Giezendanner,[43] Heiliger,[12] Krabbel,[50] Schürholz [51] and Seeliger [34]). In this connection, the case reported by Heiliger [12] has been repeatedly cited, in which the cystic dilatation was present in a stillborn fetus, born nearly at term. There was no jaundice; bile was present in the intestine, and the common duct below the dilatation was patent though considerably narrowed. Several other congenital abnormalities were present. The onset in childhood and the occurrence of symptoms early in life, or intermittently after beginning at an early age, are evidences in favor of a congenital origin. Giezendanner [43] cited many cases of frankly congenital atresia of the common duct associated with small, moderate or large cystic dilatation of the duct. In the case here reported, the thick wall of the cyst with well

developed muscle and the absence of clinical signs of obstruction are points in favor of a congenital origin. In many cases, an anatomic obstruction could not be demonstrated, and in those in which obstruction could be shown, owing to kink, valve or stenosis, the dilatation above was spherical instead of the usual cylindric dilatation observed in partial or complete obstruction due to stone, tumor or traumatic stricture. Furthermore, in spite of normal walls, the gallbladders were seldom enlarged or distended, as one would expect in ordinary obstruction, and though the hepatic ducts were described as dilated in about half the cases, the dilatation was never commensurate with that of the common duct. Valvelike folds over the mouths of the cystic and hepatic ducts may have been factors in preventing the dilatation of these structures.

2. Abnormal course of the common duct or angular insertion into the duodenum, congenital in origin, which causes kinking and obstruction (Rostowzew,[58] Arnolds,[46] Konitsky,[59] Ebner,[39] Russell[60] and Schloessmann[61]). Lavenson[45] was the first to point out that this change might be a result rather than a cause of the dilatation. Others agree with this conception, according to which the progressive enlargement pushes forward or backward, and this displacement gives rise to the abnormal course. Kolb[62] and Dreesman[20] pointed out that the condition is unlike hydronephrosis, to which it has been compared, since in the latter the ureter normally begins as a relatively large cavity, while the common duct arises by the confluence of several small ducts and is normally of uniform caliber throughout. Furthermore, it is unlike hydronephrosis in that there is not uniform dilatation of the entire system above the obstruction (Heiliger[12]). In his article, Schloessmann[61] stated that in the majority of the cases reported (1911) an abnormality of the insertion into the duodenum was present.

3. A valvelike fold of tissue over the mouth of the intraduodenal portion of the duct (Bakes,[7] Sternberg,[8] Clairmont,[56] Neugebauer[21] and Rostowzew[58]). The formation of the valve was described in 17 cases. As pointed out by Lavenson,[45] Schloessmann[61] and others,

58. Rostowzew, M. J.: Ein Fall von hochgradiger cystischer Erweiterung des Ductus choledochus, Deutsche med. Wchnschr. **28**:739 (Oct. 9) 1902.

59. Konitsky: Inaugural Dissertation; cited by Waller (footnote 17); cited by McWhorter (footnote 18).

60. Russell, R. H.: Case of Cystic Dilatation of the Common Bile Duct in a Child, Ann. Surg. **26**:692, 1897.

61. Schloessmann: Beitrag zur Kenntnis der Choledochuscysten, Deutsche Ztschr. f. Chir. **109**:160, 1911.

62. Kolb: Inaugural Dissertation; cited by Waller (footnote 17); cited by McWhorter (footnote 18).

it is hard to imagine how a valvelike fold could occur spontaneously in a cylindric structure and cause its dilatation into a cavity. Once a cavity is present, it can easily be seen that a valvelike fold might develop, as a result of sagging at the junction of the cavity with a smaller tube draining from it. The fact that such folds were present over the mouths of the hepatic and cystic ducts in many of the specimens is in favor of such an explanation. Once a valve has formed, it can be seen how it might function to cause further enlargement and obstructive symptoms. That valvular action is often present has been demonstrated in a number of cases by the presence of intermittent jaundice and pain and by fluctuations in the size of the tumor under observation.

4. Congenital narrowing of the intraduodenal portion of the duct (Heid [11]) or stenosis due to infection (Edgeworth [63]).

5. Congenital weakness of the wall of the duct. This condition was first suggested as a cause by Dreesman [20] (1908), and later by Lavenson,[45] Mayesima,[36] Neugebauer,[21] Kolb,[62] Weiss,[64] Bolle [47] and Russell.[60] Several of these authors assumed an additional factor, such as partial stenosis or occlusion due to catarrhal inflammation, spasm or edema. This whole assumption, like most of the explanations that have been offered, is not easily capable of proof. In his case, Mayesima [36] said of it, that "no other cause was present." In the cases reported in which measurements of the thickness of the wall were given, the thinnest was 2 mm.; the thickest, 7.5 mm. In many cases the wall was described as thin but tough; in only 4 was it very thin, and in 2 of these it ruptured during operative manipulation; in a few cases it was described as thick. Neugebauer [21] compared the dilatation to that of the colon in Hirschsprung's disease.

6. A complication of pregnancy. In 8 cases the symptoms bore a more or less direct relationship to pregnancy. Goldammer [29] thought that pressure from the enlarging uterus might have been a factor in his case. McWhorter [18] expressed the belief that delivery may cause a kink in the common duct due to the release of intra-abdominal pressure.

7. Primarily congenital, but with one or more of several possible modifying factors, such as infection, spasm, kink, the formation of valves or partial atresia, which cause further development of the condition (Zipf [54]).

8. Trauma. This possible etiologic factor was mentioned by Kremer.[19] In the case reported by him and by Hildebrand, the patient

63. Edgeworth, F. H.: A Case of Dilatation of the Common Bile-Duct Simulating Distension of the Gall-Bladder, Lancet **1**:1180 (May 11) 1895.

64. Weiss, S.: Ein seltener Fall von cystischen Erweiterung des Ductus choledochus, Berl. klin. Wchnschr. **46**:1843, 1909.

had a fall at the age of 7 or 8 years. At that time the abdomen was injured, and the patient herself considered that this was the cause of the symptoms.

9. Pancreatic adenomas. In his case, Budde [49] found a minute diverticulum of the ampulla of Vater and areas of fibro-adenoma in the wall of the cyst, which he demonstrated by serial section to be continuous with the pancreas. He described these areas as pancreatic adenomas. He believed that just as diverticulae of the duodenum are present in association with aberrant pancreatic tissue, so cystic dilatations or diverticulae of the common duct might be conditioned by pancreatic adenomas. In none of the other cases was fibro-adenoma found, although Reel and Burrell [38] found a few structures resembling bile ducts in the wall of the cyst in their case. They interpreted these structures as remnants of tissue of the liver and thought that the cyst might be of hepatic origin.

10. Pressure on the common duct by enlarged tuberculous mesenteric glands. This condition was suggested by Douglas.[65]

From a glance at the foregoing paragraphs it is readily seen that most authors regarded the condition as a congenital anomaly, though many others described it as idiopathic. In 4 cases in which an abdominal operation had been carried out at some time previous to the development of symptoms, the presence of the cyst was not discovered at the first operation (in the cases of Willis,[66] McWhorter,[18] Wilson [67] and Sénèque and Tailhefer [16]), but this does not necessarily mean that a cyst was not present at that time, as in each instance the first operation was performed on the lower part of the abdomen.[68]

SUMMARY

The clinical features and the pathologic changes in a case of congenital cystic dilatation of the common bile duct are reported. Eighty-two other cases reported in the literature are reviewed. The salient diagnostic features of the condition are that it occurs much more frequently in females than in males and principally in children and young adults. The three most prominent symptoms and signs are pain,

65. Douglas: Monthly J. M. Sc. (London) **14**:97, 1852; cited by McWhorter (footnote 18); cited by Dreesman (footnote 20).

66. Willis, B. C.: Congenital Cystic Dilatation of the Common Bile-Duct, Ann. Surg. **87**:48, 1928.

67. Wilson, H. V., Jr.: Choledochus Cyst, J. A. M. A. **95**:399 (Aug. 9) 1930.

68. In the case of Oglobin, not included in the present series, a young man, aged 23, was operated on for high fever, severe pain, jaundice and acholic stools of sudden onset. Nothing abnormal was found. At a second operation, undertaken for recurrence of the symptoms, a large cyst filled with clear fluid was drained. At postmortem examination, a huge cystic dilatation of the common and hepatic ducts was found.

jaundice and the presence of a tumor in the right upper quadrant of the abdomen. These changes are often intermittent over a considerable period of time. The etiology is not known, but it is generally agreeed that the condition is congenital.

From the point of view of treatment, it seems advisable on theoretical grounds to establish as early as possible an adequate communication between the biliary tract and the intestine, for usually there is a hindrance to outflow, whether from a kink, valve, fold, partial occlusion or complete atresia. A second desideratum seems to be avoidance of infection of the biliary system, which frequently occurs after its anastomosis with the intestine. As a measure against infection, excision or partial excision of the cyst to reduce stasis to a minimum would appear to be the most ideal procedure. Unfortunately, on account of technical details previously mentioned, this step is usually impossible. It must also be remembered that an adequate stoma is essential, for in one case (Hildebrand [9]) the first stoma made subsequently had to be enlarged, and in another case (Rotgans [69]) there were recurrent attacks of pain, probably due to the small stoma. On physiologic and theoretical grounds, primary anastomosis seems to be the most desirable procedure, and, in fact, it has given the best results. The condition of the patient in the late stages of complete obstructive jaundice may preclude such a procedure, so that drainage alone may be feasible. In point of fact, in cases in which the patients survived drainage long enough to permit anastomosis, the results were comparable to those in which primary anastomosis was done; on the other hand, the patients in more than half of the cases in which drainage was done died before the second stage could be carried out, and often soon after operation—from one to several days. Frequently the cause of death was hemorrhage from the fistulas in the cases reported by Ebner,[39] Ipsen,[70] Russell,[60] Seyffert,[28] Ewoyan [71] and Körte.[72]

The most encouraging finding brought out by the cases recorded in the literature is that, on the whole, the treatment adopted in the more recent cases has been better than that used in the earlier ones. The diagnosis should not be difficult if one bears in mind that congenital cystic dilatation of the common bile duct may exist, for the clinical symptoms and the physical findings are fairly characteristic. It is to be hoped, therefore, that in the future a correct preoperative diagnosis may be made more frequently, and that there will be still further improvement in the treatment and results.

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69. Rotgans, cited by McWhorter (footnote 18).

70. Ipsen, J.: Et tilfaelde af choledochuscyste, Hospitalstid. **45**:1342, 1913; cited by Waller (footnote 17).

71. Ewoyan, cited by Sebek (footnote 52).

72. Körte: Die Erkrankungen der Gallenwege, Med. Praxis, 1928, p. 117; cited by Sénèque and Tailhefer (footnote 16).

CONCRETIO CORDIS

I. A CLINICAL STUDY, WITH OBSERVATIONS ON THE VENOUS PRESSURE AND CARDIAC OUTPUT *

C. SIDNEY BURWELL, M.D.
AND
W. D. STRAYHORN, M.D.
NASHVILLE, TENN.

Disease, especially when chronic, rarely produces a single or simple change in structure or in function. Physiologic measurements of various sorts that reveal deviation from the normal when applied to patients must therefore be interpreted with caution, for it is not always easy to decide which of the mechanical or chemical changes present is responsible for the abnormal finding. From time to time, however, disease performs a simple experiment, producing a few recognizable alterations in, for example, the dynamics of the circulation: when this occurs, opportunities for study may arise that are not easily created by deliberate intention. Such an opportunity was offered by the following case.

CASE 1.—*History.*—A printer, 36 years old, white, entered Vanderbilt University Hospital through the outpatient department on Feb. 16, 1930, with the complaints of shortness of breath and generalized swelling of the body.

Previous to the onset of the present illness, the patient's health had been excellent. In childhood, he had had chickenpox, measles and whooping cough. In 1920 he had had acute gonorrheal urethritis. No history suggestive of rheumatic fever, tonsillitis, syphilis or tuberculosis could be obtained. He had had no operations.

His personal habits were good, except for the use of moderate amounts of alcoholic beverages until eight months prior to his admission to the hospital.

The patient's father died at the age of 72 of unknown immediate cause; he had suffered from cardiac and renal disease previous to this last illness. The mother died of pneumonia at the age of 67.

The onset of the present illness was six months previous to the patient's admission to the hospital. At that time he suffered repeated attacks of nausea following his usual light breakfast, but he did not vomit. The nausea was associated with attacks of cramping pain in the epigastrium lasting from thirty minutes to four or five hours, and always localized. These attacks did not occur at any other time during the day and ceased to occur after one month. Five months before admission, swelling of the feet and ankles was noticed for the first time. This would appear during the day and disappear during the night. At this time, following exposure to cold weather, the patient contracted a severe cold associated with cough and pain in the right side of his chest. Thick sputum was occasionally

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* From the Department of Medicine, Vanderbilt University School of Medicine.

coughed up, and this contained a small amount of dark blood. An examination of the lungs was made by x-rays, and the patient was told that he had a lesion in the right lung. Four and a half months before admission to the hospital, he experienced dyspnea on exertion, and about the same time he began to have attacks soon after going to bed of what he described as "smothering spells" which were relieved by sitting up. They did not occur after he got well asleep. Three months after admission the swelling of the legs became persistent, and one month later enlargement of the abdomen was noticed and also some swelling of the face, especially of the eyelids. From November 30 to January 4, the patient was treated in a hospital by rest and the administration of digitalis and showed marked symptomatic improvement, losing most of the edema. Within twelve days following discharge from the hospital, however, edema had recurred and had become worse than it had ever been. It was at this time that he was admitted to the Vanderbilt University Hospital.

Examination.—The patient was a cooperative, intelligent, well nourished white man of 36. He appeared definitely orthopneic. Respirations were shallow. The skin was slightly cyanotic. There was evidence of a generalized edema involving the chest, abdomen, back and legs, more marked in the dependent parts of the body. The cervical veins appeared greatly distended. They did not display diastolic collapse.

Examination of the head and neck showed no other abnormalities. The chest appeared broad and symmetrical, but expansion was limited. There were signs of free fluid in both pleural cavities, rising high in the axilla on the right and to the angle of the scapula on the left. A few moist râles were heard just above the level of the fluid, but the lungs were elsewhere clear. There was no "Broadbent's sign."

The cardiac impulse was barely palpable even with the patient in the left lateral position. The only visible impulse was in the third interspace 8 cm. to the left of the midsternal line. The heart did not seem enlarged to percussion. The sounds were distant. No murmurs or friction rub could be heard. The rhythm was regular, and the rate was 110. There was no demonstrable shift in position of the heart with change in position of the body. The radial pulse was small, but not quick, and was a typical paradoxical pulse with disappearance of the impulse during inspiration. The blood pressure was 118 systolic and 104 diastolic.

The abdominal wall was edematous. There was an easily demonstrable fluid wave in the moderately distended abdomen. The liver extended 8 cm. below the right costal margin. The spleen was not palpable. The results of abdominal examination were otherwise negative.

Examination of the extremities revealed nothing abnormal, except edema of the legs and thighs sufficient to exhibit deep pitting on pressure.

For the first two and a half weeks in the hospital, there was a daily rise in temperature to a point that never exceeded 101 F. and that averaged 100 F. The temperature was normal in the mornings. During the last ten days it ranged from 97 F. to 98.8 F.

The pulse rate remained elevated throughout the period of observation, ranging from 90 to 110 beats per minute. Otherwise the physical examination revealed no abnormalities.

Urinalysis revealed: a specific gravity varying from 1.010 to 1.020; an acid reaction; a slight trace of albumin, which disappeared when diuresis took place; very few cellular and granular casts and an occasional erythrocyte or leukocyte in a centrifugated specimen.

Results of the study of the blood were as follows: erythrocytes, 4,900,000; hemoglobin, 95 per cent; leukocytes, 6,000; differential count, normal; Wassermann reaction, negative; nonprotein nitrogen, 28 mg. per hundred cubic centimeters; blood sugar, 71.4 mg.; creatinine, 1 mg., and serum protein, 7.6 Gm.

A phenolsulphonphthalein renal test (with intravenous administration of dye) showed an excretion of 40 per cent of the dye during the first hour and 10 per cent during the second.

The results of fluoroscopy of the chest and of a roentgenogram of the heart taken at 7 feet (fig. 1) were reported as follows: "There is slight enlargement of the cardiac shadow to the right. It is very difficult to make out any pulsations. The outline of the heart is sharply defined, and there is apparently some pericardial effusion. The greatest diameter of the arch is 8.4 cm. The heart's shadow

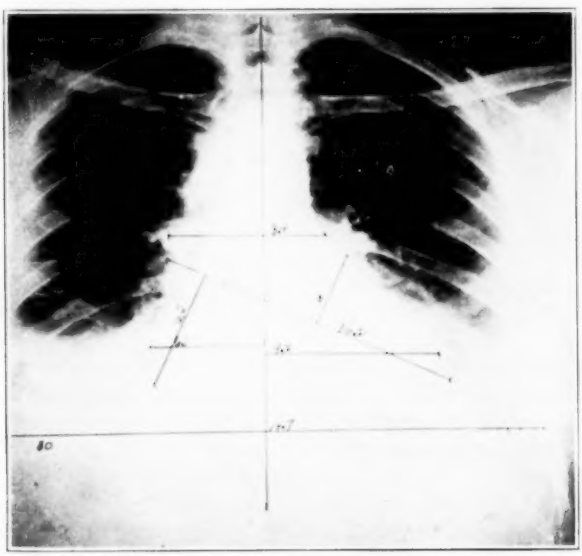

Fig. 1 (case 1).—Roentgenogram of the chest on Jan. 20, 1930, taken at a distance of 7 feet.

extends 9.2 cm. to the left and 6.1 to the right of the midline. The longest diameter of the heart is 16.2 cm. The greatest diameter of the base is 10.5 cm. The greatest diameter of the chest is 30 cm.; the distance from the midline to the left thoracic wall is 14.7 cm."

An electrocardiogram (fig. 2) showed: rate 100; P-R interval 0.16 second; inverted T waves in leads II and III; small complexes in all leads. Tracings made with the patient in different positions showed no shift of the electrical axis.

Diagnosis.—This patient exhibited many signs and suffered from many symptoms that are commonly found in patients suffering from chronic heart disease with congestive failure. It may be said that the evidences of peripheral congestion were clearer than the evidences of disease of the heart. Although there was little evidence of pulmonary edema, the patient showed marked distention of the visible veins, engorgement of the liver, peripheral edema and signs of fluid in the pleural and peritoneal cavities. The heart itself, however, showed few of the

abnormalities usually associated with congestive failure of this degree. It was but slightly enlarged, and its activity was not palpable; the sounds, far from being loud and tumultuous, were distant and weak; the pulmonic second sound was even less loud than the aortic second sound; no murmurs were heard; the systemic blood pressure and the pulse pressure were low, and the radial pulse

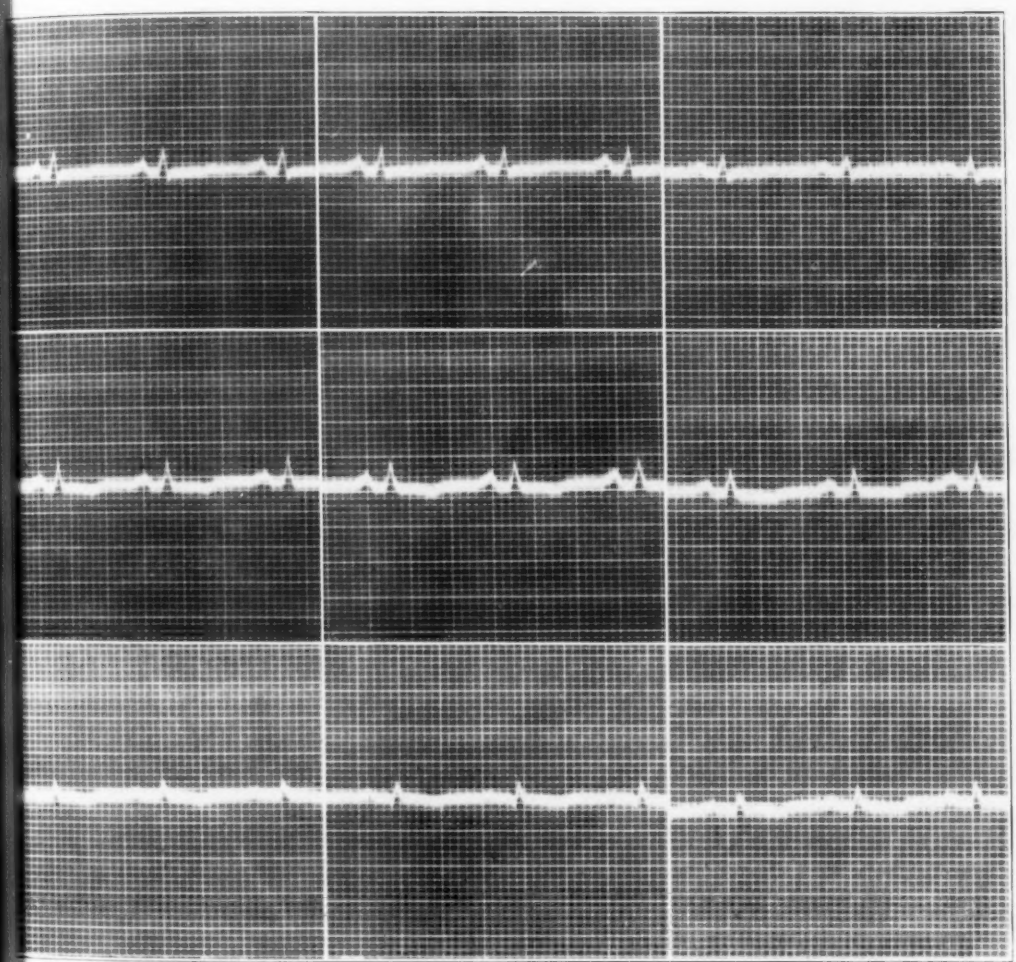

Fig. 2 (case 1).—Electrocardiographic tracings on Jan. 17, 1930, showing low voltage and slurring of QRS complexes with no shift of the electrical axis on change of position. Leads I, II and III are arranged from above downward. The horizontal columns represent dorsal, right lateral and left lateral position, respectively.

was regular, small and paradoxical. The patient thus exhibited in high degree the signs of back pressure from the right ventricle of the sort associated with failure of that ventricle. There was, however, no evidence of hypertrophy of the

right ventricle nor of any valvular or pulmonary disease to account for its failure. Moreover, the cardiac impulse was neither seen nor felt; both the cardiac dulness and the electrical axis failed to shift with change in the position of the patient, and no movement of the border of the heart was seen under the fluoroscope. Accordingly, a tentative diagnosis of adhesive pericarditis was made, of the type described by Volhard and Schmieden [1] as concretio cordis.

Following the arrival at the diagnosis of concretio cordis certain physiologic studies were projected and carried out in order to establish further the exactitude of the diagnosis, to control the treatment, and to study the mechanism of the production of symptoms. Before we embarked on these observations the patient was made much more comfortable by simple therapeutic procedures directed toward the removal of fluid. On the day of admission 1,000 cc. of fluid was removed from each side of the chest by thoracentesis, with immediate relief of the

TABLE 1.—*Determinations of the Cardiac Output of the Patient in Case 1 in the Basal, Resting Condition*

Date, 1930	Surface Area, Sq. M.	Basal Metabolic Rate, per Cent	Oxygen Consumption per Minute, Cc.	Arteriovenous Oxygen Difference, per Cent by Volume	Cardiac Output				Pulse Rate per Minute	Respirations per Min.	Blood Pressure, Mm. Hg	Pulse Pressure, Mm. Hg
					Per Minute, Liters	Per Sq. M. of Surface Area, Liters	Per 100 Cc. of Oxygen Consumed, Liters	Per Beat, Cc.				
February 1	1.71	+ 4	270	10.15	2.66	1.55	0.985	25.6	104	20	98/85	13
February 2	1.71	+ 1	246	9.04	2.72	1.59	1.10	25.6	106	22	98/86	12
February 3	1.71	0	233	11.8	1.98	1.16	0.85	18.0	110	20	96/82	14
February 5	1.71	—12	210	9.16	2.29	1.34	1.08	22.0	104	19	92/80	12
Average	1.71	— 2	240	10.04	2.41	1.41	1.00	22.8	106	20	96/83	13

orthopnea. In the succeeding three days 2,200 cc. more was removed from the right pleural cavity. Salyrgan administered intravenously, and theophylline by mouth, produced abundant diuresis. Digitalis was not given at this time. Two weeks after entry the patient had lost 7.2 Kg. and had no orthopnea, no dyspnea at rest and no edema. Furthermore, as soon as the fluid was removed from the pleural cavities it was evident that the lungs were not involved in the edema that marked the periphery. On February 1, there were no râles in the lungs, the vital capacity was 3,000 cc. (80 per cent of normal), and the oxygen saturation of the arterial blood was 98 per cent. On this evidence it was felt that diffusion in the lungs was sufficiently near to normal for studies of the cardiac output to be undertaken. These studies were carried out by the acetylene method of Grollman.[2] First, four determinations were made under "standard basal" conditions. The results of the studies are recorded in table 1. The determination of the cardiac output by the method of Grollman gives results that vary somewhat from those obtained by other methods. Therefore, in table 2, for convenience in comparison,

1. Volhard and Schmieden: Ueber Erkennung und Behandlung der Umklammerung des Herzens durch schwielige Perikarditis, Klin. Wchnschr. **2**:5, 1923.

2. Grollman, A.: The Determination of the Cardiac Output of Man by the Use of Acetylene, Am. J. Physiol. **88**:432, 1929.

we summarized the results of Grollman's [3] studies of normal persons and added also the results of studies made by us on a small series of normal persons, using their method. Certain of the figures are compared graphically in figure 3.

Scrutiny of these data shows definite departure from the normal on the part of the patient. His cardiac rate, in spite of careful attention to the details of the "standard basal" conditions, never fell below 104 and averaged 106. The total

TABLE 2.—*Comparison of the Averages of the Determinations of the Cardiac Output of Forty Normal Subjects in Grollman's Series and of Five Normal Subjects in Our Series*

	Surface Area, Sq. M.	Oxygen Consumption per Minute, Cc.	Arteriovenous Oxygen Difference, per Cent by Volume	Cardiac Output				Pulse Rate per Minute	Pulse Pressure, Mm. Hg
				Per Minute, Liters	Per Sq. M. of Surface Area, Liters	Per 100 Cc. of Oxygen Consumed, Liters	Per Beat, Cc.		
Average of Grollman's series of 40 normal subjects	1.76	230	5.93	3.88	2.21	1.68	60	64	38
Average of our series of 5 normal subjects	1.98	241	6.38	3.78	1.91	1.56	58	65	32

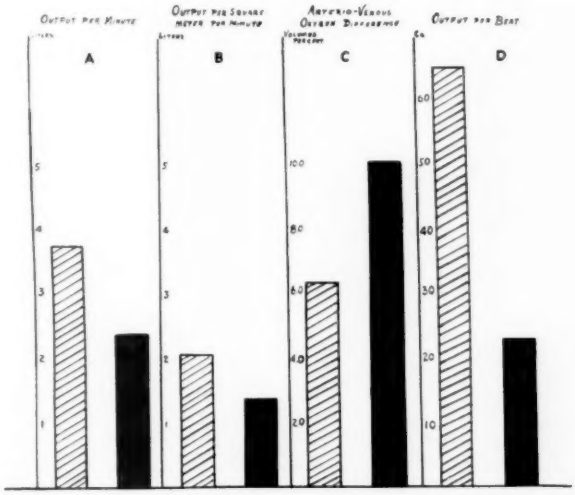

Fig. 3 (case 1).—Graphic comparison of the results of determinations of the cardiac output per minute (*A*), the cardiac output per square meter (*B*), the arteriovenous oxygen difference (*C*) and the cardiac output per beat (*D*) of a normal control subject (shaded columns) and of the patient (solid columns).

consumption of oxygen was not abnormal, but the oxygen taken out of each liter of blood as it passed through the body reached the high average of 101 cc.

3. Grollman, A.: Physiological Variations in the Cardiac Output of Man: VI. The Value of the Cardiac Output of the Normal Individual in the Basal Resting Condition, Am. J. Physiol. **90**:210, 1929.

(10.1 per cent by volume), as compared with average normal figures of 59 and 64 cc. The cardiac output per minute was lower than in any normal person studied and 36 per cent lower than the average reported by Grollman.[2] The validity of the difference is shown by the fact that it holds also for the cardiac output per square meter of surface area and per hundred cubic centimeters of oxygen absorbed.

The greatest and most significant difference between this patient and normal persons is seen in the figures for the output of his heart per beat. The output per beat of normal resting persons varies between 55 and 80 cc. and averages about 65 cc. The output per beat of the patient varied between 18 and 26 cc. This finding is to be correlated with the low pulse pressures that were recorded on the occasion of each observation.

The venous pressure in concretio cordis was measured by Volhard and Schmieden and found to be increased to as much as 300 mm. of water. During the progress of our studies, determinations were made of the venous pressure by the method of Moritz and von Tabora.[4] With the needle in the right basilic vein, the venous pressure measured 240 mm. of water. During gentle flexion and extension of the left arm, the venous pressure rose steadily. When the arm was again at rest, the pressure fell slowly to its previous level of 240 mm.

When the identical method was applied to a normal person, his resting pressure was found to be 70 mm. Flexion and extension of the left arm had no effect on the level of pressure in the vein of the right antecubital space.

The evidence to this point indicated a backing up from the right side of the heart due either to obstruction in the great veins or to a fibrous coat around the heart sufficiently thick and tight to prevent a proper diastolic relaxation and filling. The appearance of the heart in the fluoroscope was in favor of the latter view. It was felt that a decisive test would be the response of the heart to exercise. If the fibrous envelop assumed to be surrounding the heart was present, it was to be expected that the output of the heart per beat could not increase in response to demand for an increased circulation. Accordingly the output of the heart per minute and per beat was determined during exercise sufficient to increase the consumption of oxygen from an average of 240 cc. when the patient was at rest to an average of 535 cc. per minute. For purposes of comparison, similar observations were made on a normal young man. The observations made during exercise are summarized in table 3 and compared graphically in figure 4.

It is seen that in the normal man both the pulse rate and the heart's output per beat were increased. The cardiac output per minute was thus increased about as much as the consumption of oxygen. The utilization of oxygen remained about the same, indicating that the oxygen tension in the tissues was not greatly altered. The normal subject felt no fatigue and no dyspnea. In the patient, the circulatory response to exercise was quite different. His pulse rate, already 106, increased to 144, but the output per beat remained almost exactly at the level that was observed when he was resting. The total cardiac output per minute increased much less than the consumption of oxygen. The utilization of oxygen rose to 130 cc. per liter (77 per cent of the total oxygen capacity); the oxygen tension of the

4. Moritz, F., and von Tabora, D.: Ueber eine Methode beim Menschen den Druck in oberflächlichen Venen exakt zu bestimmen, Deutsches Arch. f. klin. Med. **98**:425, 1910.

TABLE 3.—*Determination of Cardiac Output Under Basal Conditions and Following Exercise on a Normal Control Subject and on a Patient with Concretio Cordis (Case 1)*

	Surface Area, Sq. M.	Basal Metabolic Rate, per Cent	Oxygen Consumption per Minute, Cc.	Arteriovenous Oxygen Difference, per Cent by Volume	Cardiac Output				Pulse Rate per Minute	Respirations per Min.	Blood Pressure, Mm. Hg	Pulse Pressure, Mm. Hg
					Per Minute, Liters	Per Sq. M. of Surface Area, Liters	Per 100 Cc. of Oxygen Consumed, Liters	Per Beat, Cc.				
Normal control under basal conditions....	1.84	— 7	234	6.83	3.42	1.87	1.46	50	69	11	90/62	32
Normal control after exercise	1.84	+178	683	7.12	9.60	5.22	1.40	80	120	24	125/85	40
Patient under basal conditions (av. of 4 determinations).....	1.71	— 2	240	10.04	2.41	1.41	1.00	23	106	20	96/83	13
Patient after exercise.....	1.71	+140	535	13.00	4.12	2.41	0.77	29	144	38	100/88	12

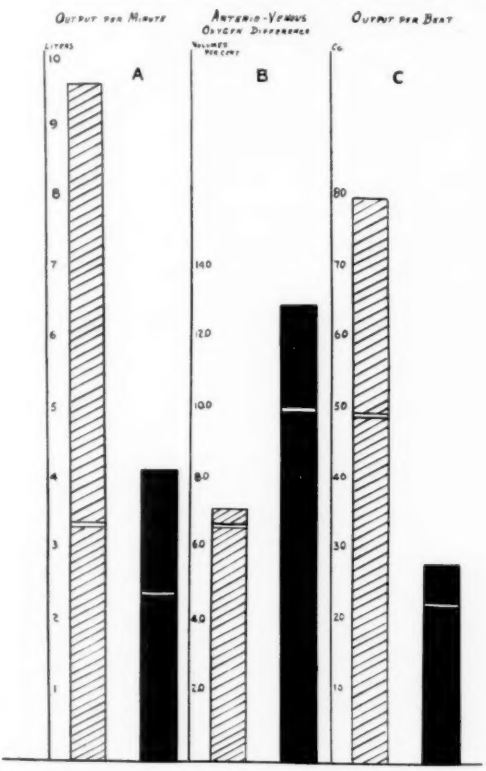

Fig. 4 (case 1).—Graphic comparison of the results of determinations of the cardiac output per minute (*A*), the arteriovenous oxygen difference (*B*) and the cardiac output per beat (*C*) of a normal control subject (shaded columns) and of the patient (solid columns) before and after exercise. The amount of change with exercise is shown by the height of the columns above the horizontal lines.

tissues was therefore lower, and the patient felt exhausted and short of breath. His pulse pressure remained at 12 mm. of mercury; that of the normal control rose from a resting value of 28 mm. to 40 mm. It was felt that this experiment substantiated our diagnosis of concretio cordis, because it offered evidence of the inability of the heart to dilate to a normal extent.

Treatment.—When we speculated as to the effect of digitalis in this case it was thought likely that it would have no beneficial effect. If the rate of 106 when the heart was at rest represented, as seemed probable, an increase compensating for the small volume per stroke, slowing of the heart by digitalis would diminish the cardiac output per minute still further and thereby increase the severity of the symptoms. Digitalis decreases the output of the heart per minute in normal dogs [5] and in normal men.[6] If the same effect resulted in this patient who already had an abnormally low output of the heart per minute, the influence would be for the worse. He gave a history of having been relieved by digitalis combined with rest during an earlier period of his illness. Accordingly digitalis was given, but with caution. After 1.6 Gm. of the leaf had been administered in three days, the patient complained of increasing dyspnea and distress. Two determinations of the cardiac output were made. The output of the heart per minute was reduced only slightly, and the decrease was mainly accounted for by a fall in the basal metabolic rate. The basal cardiac rate was 90, or 16 beats less than the previous average. This association of digitalization and slowing of the heart with increase in dyspnea and edema serves still further to emphasize the difference between this patient's condition and true heart failure.

The nature of the defect being thus established, the questions of prognosis and further treatment arose. Granting the accuracy of the diagnosis, it was clear that the patient's disability was severe, that it could be expected to grow worse rather than better, and that treatment by any method except mechanical relief would be in vain. If, however, enough of the confining coat of fibrous tissue could be removed to permit the heart increased freedom in relaxation, improvement might be expected. The attempt was made. The procedure is described by the operator in a second paper.

The patient died suddenly about twelve hours after operation.

Postmortem Observations.—The chief observations made at postmortem examination follow:

When the wall of the chest was opened, the heart was found to be small, and it was comparatively free from adhesions to the mediastinum, diaphragm and wall of the chest. The visceral pericardium and the parietal pericardium were closely fused throughout by fairly friable fibrous adhesions, but these could be separated by the finger. When the pericardium was opened, the parietal layer was found to measure from 2 to 3 mm. and the visceral layer from 1.5 to 2 mm. in thickness. Together they formed an inextensible fibrous capsule around the heart, as may be seen in figures 5 and 6. On cut section, the heart showed extreme atrophy, with brown, very friable muscular fibers. The endocardium was smooth and velvety, and there was no evidence of valvular disease. There was definite chronic passive congestion of the liver, spleen and kidneys, but not of the lungs.

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5. Harrison, T. R., and Leonard, B. W.: The Effect of Digitalis on the Cardiac Output of Dogs and Its Bearing on the Action of the Drug in Heart Disease, J. Clin. Investigation **3**:1, 1926.

6. Burwell, C. S.; Neighbors, DeWitt, and Regen, E. M.: The Effect of Digitalis upon the Output of the Heart of Normal Men, J. Clin. Investigation **5**:125, 1927.

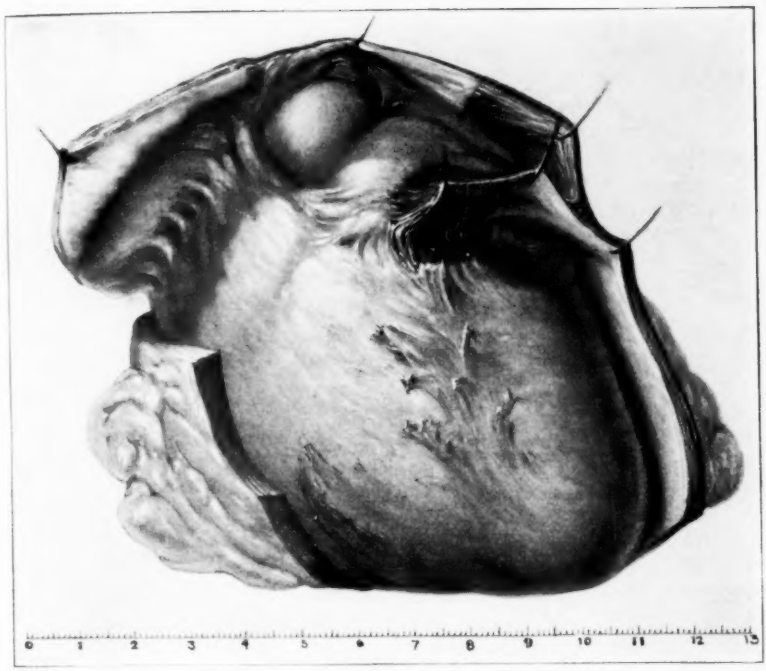

Fig. 5 (case 1).—Drawing of exterior surface of the heart of the patient, showing obliteration of the surface landmarks by the thickened epicardium, and the cut edge of the enormously thickened pericardium. The thinness of the muscular wall is shown by the tear in the region of the right ventricle.

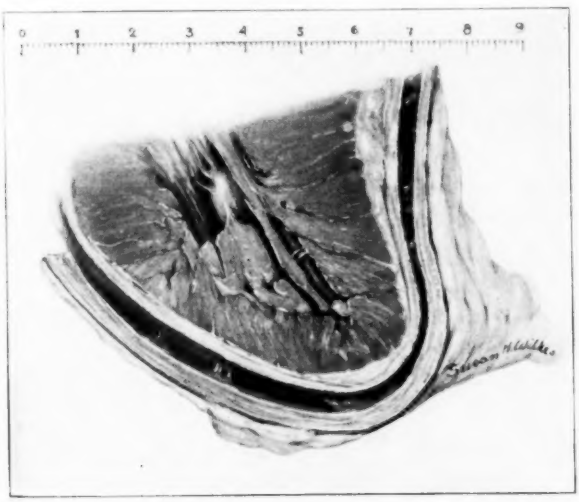

Fig. 6 (case 1).—Drawing of the cut surface of the heart of the patient, showing the small size, the atrophy of the cardiac muscle and the markedly thickened epicardium and pericardium.

In the microscopic examination of sections of the heart the thickened sections of both parietal and visceral pericardium showed evidence of chronic progressive proliferative tuberculous infection. Giant cells, small areas of caseation and an infiltration by mononuclears and lymphocytes were scattered diffusely throughout the sections.

This case, then, was one of tuberculous pericarditis resulting in the encompassment of the heart by a thick and closely applied layer of firm fibrous tissue. Experimental observations demonstrated certain deviations from normal in the functioning of the patient's heart. These deviations served to establish the diagnosis and also to supply a rational explanation of the symptoms and signs.

TABLE 4.—*Determinations of the Cardiac Output of the Patient in Case 1 Under "Basal" Conditions Before and After Digitalization*

Date and Conditions of Experiment	Surface Area, Sq. M.	Basal Metabolic Rate, per Cent	Oxygen Consumption per Minute, Cc.	Arteriovenous Oxygen Difference, per Cent by Volume	Cardiac Output				Pulse Rate per Minute	Respirations per Min.	Blood Pressure, Mm. Hg	Pulse Pressure, Mm. Hg
					Per Minute, Liters	Per Sq. M. of Surface Area, Liters	Per 100 Cc. of Oxygen Consumed, Liters	Per Beat, Cc.				
Average of 4 determinations under basal conditions before digitalization	1.71	— 2	240	10.04	2.41	1.41	1.00	22.8	106	20	96/83	13
Feb. 11, 1930, determination under basal conditions after 1.5 Gm. digitalis folia in 3 days	1.71	—10	212	9.10	2.33	1.36	1.10	25.0	93	17	94/84	10
Feb. 12, 1930, determination under basal conditions after 1.6 Gm. digitalis folia	1.71	—18	197	10.8	1.83	1.09	0.93	20.3	90	17	98/88	10

The essential defect, as revealed by these studies, was the limitation of the diastolic relaxation of the heart by the encircling scar tissue and the consequent fixation of the output per beat at an abnormally low level. This limitation of the output per beat made it impossible for the output of the heart per minute to increase except so far as this could be brought about by increase in the already rapid cardiac rate. The dyspnea that the patient experienced on slight exertion was presumably due to the inability of the heart to increase its output adequately, and thus resembled the dyspnea suffered by normal persons after severe exertion. The edema, however, was not due to the diminished cardiac output, but, certainly in the main, to the elevated venous pressure. That this is so is indicated by the distribution of the edema in the patient's body. The volume of blood flow per unit of time was necessarily diminished in both the peripheral and the pulmonary areas, but congestion

and edema were observed only in the area drained by the systemic veins in which the pressure is known to have been elevated far above the limits of normal. This is important enough to be restated: In the presence of diminished flow of blood through the lungs there were no physical signs of pulmonary edema during life nor any visible passive congestion in the lungs after death; massive edema occurred in the parts of the body in which the pressure in the veins was increased. This observation is of theoretical importance, since it tends to show that edema, a chief manifestation of heart failure, is more dependent on increased venous pressure (" back-pressure") than on alteration in the volume flow of blood.

Eyster [7] emphasized the parallelism existing between the height of the venous pressure and the severity of the manifestations of cardiac failure. The fact that many symptoms and signs are common to this type of adhesive pericarditis (concretio cordis), on the one hand, and to congestive heart failure, on the other, may be explained by the existence of an abnormally high venous pressure in both groups of cases. The differences between the clinical manifestations of concretio cordis and those of congestive failure indicate that the increase of venous pressure may be brought about by different mechanisms.

Another patient with concretio cordis was the subject of similar studies, carried out some months after a successful decortication of the heart.

CASE 2.—*History.*—A 25 year old Negro entered Vanderbilt University Hospital on May 28, 1929, with a chief complaint of swelling of the abdomen.

The past history revealed good general health previous to the present illness. The patient had had measles, mumps, whooping cough and smallpox in childhood and pneumonia in 1922. He had received a stab wound in the right side of the chest near the upper border of the sternum five months previous to admission to the hospital. This had healed without any complications.

Except for the consumption of about 1 pint (about 250 cc.) of whisky a week during the past two years, the patient's personal habits had been good.

Three brothers were born dead, and a sister died soon after birth. The family history was otherwise unimportant.

The present illness began five months before admission to the hospital, when the patient noticed moderate swelling of his face on awakening in the morning. This would disappear in a few hours and after three weeks ceased to occur. During the latter part of December, 1928, swelling of the abdomen and legs was noted. This progressed rapidly and soon forced him to stop work. After two months he went to a hospital where he stayed six weeks and improved, the swelling of the abdomen and legs disappearing. Three weeks following his discharge this swelling reappeared and became persistent. There was moderate dyspnea on exertion, but no orthopnea and no cough. There had been slight transient precordial pains during the course of his illness.

Examination.—The patient was an intelligent Negro. He appeared moderately short of breath on exertion, showed evidence of marked weakness, and had slight

7. Eyster, J. A. E.: The Clinical Aspects of Venous Pressure, New York, The Macmillan Company, 1929.

fever with a daily swing from 98 to 100 F. There was a slight general glandular enlargement.

Examination of the head did not disclose any abnormality. The neck showed striking venous pulsations with bilateral distention of the veins.

There was evidence of hydrothorax on the right side. No cardiac impulses were palpable. The area of cardiac dulness was not enlarged. The sounds were distant, and no murmurs or friction rub could be heard. There was no demonstrable shift in the position of the heart with change in the position of the body. The rhythm was regular, the rate 100 and the blood pressure 110 systolic and 80 diastolic.

The abdomen was distended with fluid, and the liver was markedly enlarged. There was pitting edema of the extremities.

TABLE 5.—*Determinations of Cardiac Output of the Patient in Case 2 Under Basal Conditions and Following Exercise Twelve Months Following Operation for Concretio Cordis*

Date and Conditions of Experiment	Surface Area, Sq. M.	Basal Metabolic Rate, per Cent	Oxygen Consumption per Minute, Cc.	Arteriovenous Oxygen Difference, per Cent by Volume	Cardiac Output				Pulse Rate per Minute	Respirations per Min.	Blood Pressure, Mm. Hg	Pulse Pressure, Mm. Hg
					Per Minute, Liters	Per Sq. M. of Surface Area, Liters	Per 100 Cc. of Oxygen Consumed, Liters	Per Beat, Cc.				
May 21, 1930, under basal conditions	1.61	+ 1	219	6.29	3.48	2.16	1.59	43	80	22	90/55	35
May 23, 1930, under basal conditions	1.61	+ 1	220	6.11	3.6	2.24	1.64	46	79	18	85/48	37
June 4, 1930, after exercise	1.61	+212	670	8.77	7.64	4.73	1.31	68	112	40	110/78	32
June 6, 1930, after exercise	1.61	+212	678	8.37	8.10	5.02	1.19	81	100	34	112/78	34

The results of examination were otherwise negative.

The urine was normal, except for a trace of albumin on one occasion. The cells of the blood were normal. The Wassermann reaction was negative.

A phenolsulphonphthalein renal test with intravenous administration of dye showed an excretion of 80 per cent of the dye in two hours.

An x-ray picture of the chest showed increased density over the right lower lobe, suggesting a small amount of fluid. No evidence of calcification of the pericardium could be made out.

An electrocardiogram showed a rate of 100, a P-R interval of 0.16 second and inverted T waves in all leads. Tracings made with the patient in different positions showed very slight shift of the electrical axis.

In view of the findings, a diagnosis of concretio cordis was made, and because of the patient's complete disability an operation was advised.

The operation and the patient's subsequent course are discussed in a second paper. It was during his third admission to the hospital, in June, 1930, that studies on the cardiac output were made.

Observations of venous pressure and cardiac output were not made in this case before operation, but similarity in the manifestations of

the two cases make it certain that the venous pressure was high and probable that the cardiac output per minute and per beat was diminished. Twelve months after operation these studies were carried out, at a time when the patient had no edema, no dyspnea and no enlargement of the liver. As may be seen in table 5, the results show no essential deviation from the average normal values. The venous pressure was within normal limits.

Therefore, it can be said that in one patient with concretio cordis and congestion the venous pressure was high and the cardiac output per minute and per beat was low, while in another patient successfully operated on for a similar condition there was no congestion and the venous pressure and the output per beat were within the limits found in normal subjects.

Since the writing of this paper there has appeared a report of the impressive work of Beck and Griswold.[8] These observers were able to produce experimentally in dogs "fibrosis and contracture of the parietal pericardium, or epicardium, or both, forming a casing of scar which compresses the heart and primarily obstructs its filling . . . ." The development and contracture of the scar tissue were accompanied by a rise in venous pressure and by ascites, hydrothorax and edema, pulmonary and subcutaneous; the pulse became small and rapid, and the output of the heart per minute was decreased. Resection of the scar was followed, in general, by a fall in the venous pressure toward the normal level, by a diminution or disappearance of edema and by an increase in the output of the heart per minute. After operation, in most cases the heart was larger than before. These authors also reported a case similar in type to the two reported here. Their patient exhibited, before operation, a high venous pressure, a rapid pulse and a small pulse pressure. After pericardiectomy the venous pressure and the pulse rate fell, and the pulse pressure rose to twice its former value.

The observations of Beck and Griswold on dogs seem both to confirm and to be confirmed by our studies on patients, particularly in regard to the high venous pressure and the diminished output of the heart.

SUMMARY

In a patient with concretio cordis studies of the output of the heart and of the venous and arterial pressures seemed to indicate that the essential defect in the circulatory mechanism was the failure of the heart to relax (dilate) to the normal extent. Similar studies of a patient successfully treated by operation for a similar condition revealed no departure from normal.

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8. Beck, C. S., and Griswold, R. A.: Pericardiectomy in the Treatment of the Pick Syndrome, Arch. Surg. **21**:1064 (Dec., pt. 2) 1930.

PLASTIC OPERATION FOR ANAL INCONTINENCE

FURTHER REPORT *

HARVEY B. STONE, M.D.
BALTIMORE

Within the last three years, Wreden [1] and I [2] described a new method of restitution of anal control following damage or loss of the sphincter muscle. At that time Wreden reported one successful case, and I reported one successful and one unsuccessful case; since then Ransohoff [3] has reported an additional case in which this method was successful. My purpose in the present paper is to record five other cases, to call attention again to this valuable principle of treatment in a most discouraging situation and to introduce some further modifications of technic which seem to be improvements. The essential principle of Wreden's operation is to harness the two gluteus maximus muscles by slings of fascia that pass around the anal canal, in such a manner that when the glutei contract these fascial slings will be drawn taut. As the slings from the two sides interlock, this tightening closes the anal canal and gives the patient voluntary control over the closure of the lower end of the bowel.

REPORT OF CASES

The case reports, condensed to essentials, follow.

CASE 1.—Mrs. E. M., white, aged 44, was admitted to the Church Home and Infirmary, with the complaint of diarrhea and incontinence of feces. She had a long surgical history of twelve operations, including removal of a ruptured appendix, removal of the gallbladder, cesarean section, hysterectomy, perineal repair and several rectal operations. Three years previous to her admission for the present illness, the sphincter muscle had been divided for an ulceration of the rectum. Since that time she had had no control over the bowels in spite of two or three attempts at repair of the sphincter. Eighteen months before admission diarrhea developed with from twenty to forty stools daily. Parasites were not found on repeated search, and ulceration was not seen. A diagnosis of mucous colitis was made.

On examination, the patient was found to be a highly nervous woman with multiple scars and an extremely annoying mucous colitis. The anal outlet was patulous and showed considerable scarring. On palpation, no sphincteral tone

* Submitted for publication, Dec. 26, 1930.

1. Wreden, R. R.: A Method of Reconstructing a Voluntary Sphincter Ani, Arch. Surg. **18**:841 (March) 1929.

2. Stone, H. B.: Plastic Operation for Anal Incontinence, Arch. Surg. **18**: 845 (March) 1929.

3. Ransohoff, J. Louis: Ann. Surg. **90**:317, 1929.

could be felt, and when the patient was asked to contract the anal outlet voluntarily, visible contraction did not take place.

On Oct. 18, 1929, an operation was done with the technic described in my previous article, except that a piece of fascia from the patient's thigh was used to form the strips. The wound became infected to a considerable extent, and some of the fascia sloughed out. It finally healed, and the patient was discharged six weeks after operation. At this time the mucous colitis was greatly improved, and there was distinct contracting power in the anal canal when the gluteus muscles were tightened. A report one year later showed much improvement over her previous condition, and her control was considered about 85 per cent. This case can be classed as one of definite improvement.

Case 2.—L. deG., a girl, white, aged 16, was admitted to the Church Home and Infirmary. She had a spina bifida with paralysis of many of the muscles in both legs below the knee with pes equinocavus and with incontinence of the rectum and of the bladder; this condition had existed since birth and was clearly due to a developmental fault in the lower part of the spine, with extensive disturbance of the nerves.

On Jan. 14, 1930, operation was performed, the strips being of prepared fascia and not autogenous. The wound healed practically per primam except for a slight serous drainage. The patient was discharged on January 28, and at that time the result of the operation was considered to be very good. She has been seen since that time, and as she had learned to utilize the gluteus muscles better, her improvement has continued. The patient considers the result to be practically perfect.

Case 3.—H. H., white, a man, aged 63, was admitted to the Church Home and Infirmary. In September, 1928, he was operated on for an extensive anal fistula. In the course of this operation it was necessary to divide the sphincter muscle. The fistula healed, but following recovery from the operation there was considerable incontinence but not complete loss of anal control. This weakness of control was quite annoying, particularly as this patient also had a mucous colitis of several years' standing and was unable to retain mucous stools at times.

Operation was done on March 25, 1930, the prepared fascia being used for the strips. He was discharged on April 7. The wound healed practically per primam, there being only a slight amount of serous drainage. The man's condition was greatly improved. When he was seen again three months later, the result was satisfactory but not perfect. The only difficulty that the patient has is that there is some slight seepage of moisture unless he is careful to keep his glutei tense at times. This occurs only when one of the mucous bowel movements is present in the rectum. He does not have any difficulty controlling actual stool nor any difficulty with gas.

Case 4.—Mrs. J. O., white, a woman, aged 37, was admitted to the Union Memorial Hospital on March 16, 1930, with a diagnosis of ovarian cyst, uterine myomas and also of relaxed sphincter ani. Operation was done on April 22, the uterus and the right adnexa being removed. Convalescence from this operation was satisfactory, and on April 19 it was decided to attempt a plastic operation for the anal incontinence with strips of fascia. This incontinence followed forceps delivery in 1912; in 1913 an unsuccessful attempt was made to repair the damage by the usual plastic operation on the perineum. A second unsuccessful attempt at sphincter repair was made in 1921. The incontinence had continued without improvement and had recently become worse. The operation on April 19, 1930, was performed by Dr. J. Arthur Weinberg, at that time resident surgeon of the

Union Memorial Hospital. Following the operation, the wounds became slightly infected and drained for two or three weeks before finally closing. There was also some sloughing of the distal end of the rectal mucosa, which was probably due to some strangulation by the bands of fascia. The patient was discharged from the hospital on May 11 with the wound entirely healed; she had good control of the bowel movements but not complete control of gas. The sphincter at that time admitted the index finger. She was reexamined on Oct. 24, 1930, when the following note was made: "Sphincter tone good. Admits index finger. Wounds entirely healed. Patient has complete control except when she becomes very nervous and excited. Bowels move regularly. No soiling whatever. Result considered perfect."

CASE 5.—C. B., white, aged 32, was operated on in 1928 and again in 1929 for a rectal fistula. Since that time he had been troubled with discharge and itching about the anus and was unable to control his bowel movements after taking laxatives. He was first given an injection of alcohol for the pruritus, but he returned later complaining of incomplete control of the bowel. On rectal examination, an inflamed area was seen about the anus with a mucoid discharge coming from the rectum. The rectal sphincter was relaxed, and the patient apparently had very little control of it. Operation was performed on March 4, 1930, at the Church Home and Infirmary by Dr. Ervin B. Wallace, at that time resident surgeon at the Church Home. The material used for the strips was prepared fascia. Recovery was uneventful, and the wounds healed by first intention. The discharge from the rectum was considerably lessened. Sphincter control improved so much that the patient could now hold an enema. He was discharged from the hospital in two weeks and seen again in the middle of October, 1930, six months after the operation. He still had some pruritus in the skin about the anus, but his sphincter was working perfectly. Dr. Wallace reports an entirely satisfactory result. In this instance the patient was distinctly subnormal mentally, and it was felt that because of his lack of intelligence he did not utilize his gluteus muscles to the best possible advantage.

Summary.—In the five cases reported, together with the four others reported in the articles previously referred to, the results were as follows: successful, five cases; practically successful, two cases; decidedly benefited, one case, and failure, one case. In the two in which the results were the least satisfactory, infection was much more pronounced than in any of the other cases, and it was felt to be without doubt the cause of failure. The desire to reduce the possibility of infection in a field where strict aseptic postoperative conditions are almost impossible has led to the modification of the operation in some details, and at present the following procedure is employed.

OPERATIVE PROCEDURE

With the patient in lithotomy position, after as careful cleansing of the field as possible, including cleansing enemas for two days previous to operation, two symmetrical incisions are made, one on each side, about 4 cm. lateral to the anal margin and slightly posterior to it. These incisions slant somewhat laterally toward their anterior ends, are about 2 cm. long and are carried well into the subcutaneous fat. After the two incisions have been made, a closed long curved

clamp (Kelly) is introduced through one incision and by blunt dissection is forced around through the subcutaneous tissues in front of the anus, and its tip is made to emerge into the other incision. The clamp is then opened and grasps the ends of two strips of fascia, 0.5 cm. wide and from 22 to 25 cm. long. This fascia may be either autogenous from the patient's fascia lata or prepared by the Koontz method. The clamp grasping the strips of fascia is then withdrawn the way it

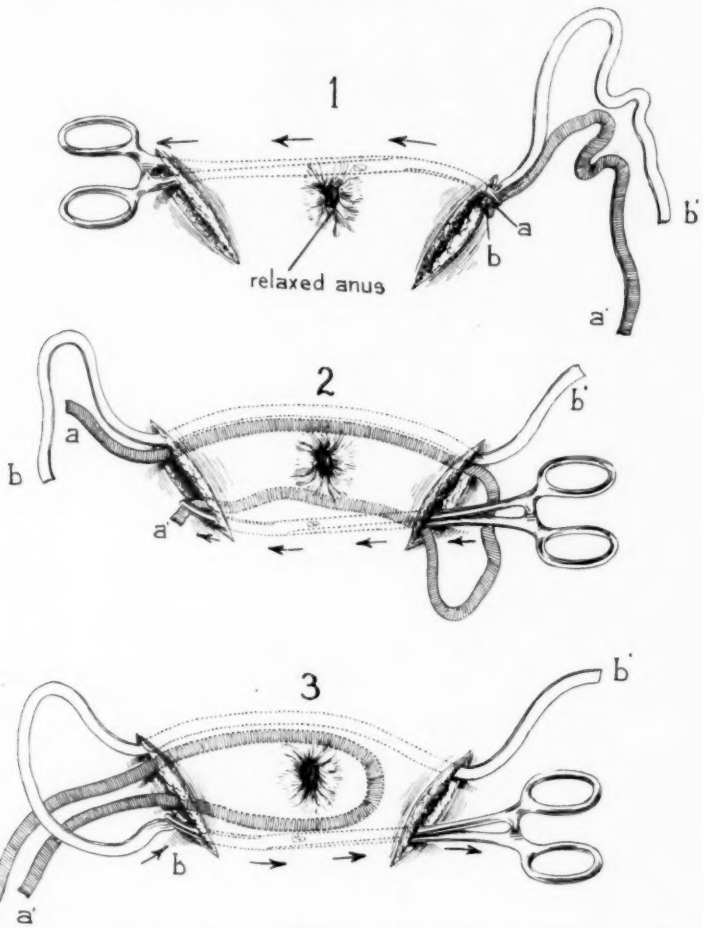

Fig. 1.—Steps in the plastic operation for anal incontinence: *1*, clamps are passed subcutaneously in front of the anus grasping the strips of fascia and are about to be pulled from one side to the other; *2*, both strips are passed subcutaneously in front of the anus and one posteriorly; *3*, the loops of fascia encircle the anus subcutaneously in opposite directions.

was introduced. This threads both strips of fascia in front of the anus. A second Kelly clamp now grasps the end of one of the strips, is passed again into one of the incisions and is forced to burrow its way subcutaneously to the other incision, this time behind the anal canal. As it emerges into the opposite wound,

the strip of fascia clamped in its tip is seized and more of it drawn through the wound. This strip now encircles the anus subcutaneously, entering at one incision, and its opposite end emerging from the same incision. The clamp, which meanwhile has remained in its posterior tunnel, is opened, grasps the free end of the second strip of fascia and is pulled back the way it was pushed in. It brings back with it the end of the second strip of fascia which now also encircles the anus subcutaneously, but in the opposite direction to the first strip, each forming

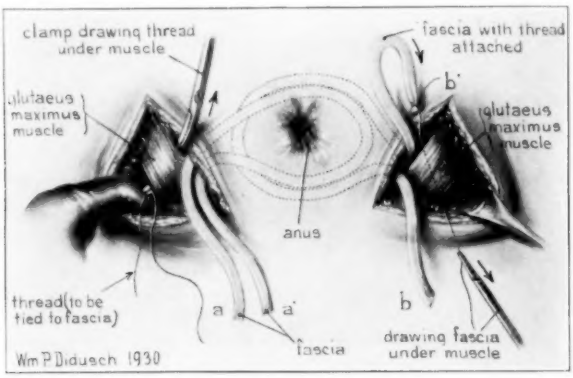

Fig. 2.—The outer ends of the strips of fascia are passed around the bundles of the gluteus muscle.

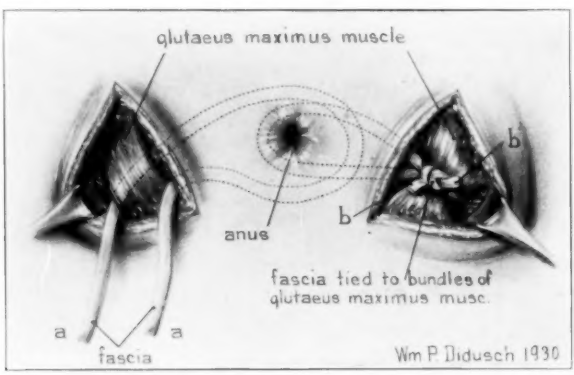

Fig. 3.—The loops of fascia are tied on the right side, ready for tying on the left.

a loop with the free ends of the same strip protruding through the same incision. From one of the incisions, blunt dissection is now carried outward and backward fairly deep in the fat until the mesial edge of the gluteus maximus is reached and identified. A bundle of fibers of this muscle about as thick as one's index finger is now surrounded by one end of the strip of fascia protruding from the incision. This may be done easiest by carrying a linen thread in an aneurysm needle around the muscle bundle and using the thread as a tractor, tied to the end of the strip of fascia, to pull the strip around the muscle bundle. This end of the strip is

now brought back into the incision and tied tightly to its own other end, thus forming a closed loop, one end of which encircles the anal canal and the other encircles a bundle of gluteus muscle. Exactly the same steps are executed in the other incision with the two ends of the other strip of fascia. The knotted ends of the fascia sink into the depths of the wounds, and the skin is closed as the operator may prefer. The two small incisions are sealed with some impervious dressing, and the bowels are kept tied up for from seven to ten days following the operation.

The anus is now encircled by two loops of fascia, running off into opposite directions, tied under some tension about bundles of gluteus muscles. When the glutei are voluntarily contracted they will pull the loops still tighter and give a mechanism for voluntary increase in closing pressure on the anal canal.

As has been said, the principle of this operation is that of Wreden. The method differs from his and from my own first method by reducing the number of incisions from four to two and by moving these two further from the anal orifice. The mechanics of the operation remain the same, but the chance of avoiding infection of the wound is improved. The results in the last four cases herewith reported, in which this newer modification was used, have been very satisfactory. If infection can be avoided, the percentage of good results should be high.

There are several points of interest in regard to this operation that deserve a further word. The operation depends on the utilization of the glutei muscles. It is obvious that if these muscles for any reason are not functioning, this operation cannot be expected to succeed. Also, until the patient learns to use the glutei properly, the full benefit is not obtained. Hence education of the patient in this regard is important. He must be trained to contract the buttocks when necessary. It follows that patients who cannot be taught to do this, because of lack of interest or intelligence, do not improve as much as others. The last case reported was an instance in point. The anatomic and surgical result was excellent, but the patient did not always remember to use his new power of anal control. Because of this necessity for education, the patients in successful cases do not show as much improvement immediately as they will ultimately.

SUMMARY

Five further cases of anal plastic operations are reported and reviewed with four previously recorded cases; the results in five were excellent, in two good and in two unsatisfactory. A further modification of the method to secure better protection against infection is described.

PEPTIC ULCER IN EXPERIMENTAL OBSTRUCTIVE JAUNDICE*

JESSE L. BOLLMAN, M.D.
AND
FRANK C. MANN, M.D.
ROCHESTER, MINN.

During the course of our studies on the experimental control of ascites, extending over several years, we have had occasion to observe a large number of dogs with complete obstructive jaundice. Many of these animals were lost to the original study because of the early development of perforated duodenal and gastric ulcers. Only a few studies have been made concerning ulcer complicating complete obstructive jaundice. Of the recent work bearing directly on the subject, Berg and Jobling[1] (1930) found that in three of five animals, peptic ulcers developed following complete obstructive jaundice. In one dog multiple gastric erosions were encountered forty-seven days after ligation of the common bile duct. One hundred and eight days after operation, another animal had two perforated chronic duodenal ulcers, and the third dog had a single chronic duodenal ulcer after one hundred and eighty-nine days. These authors also often found peptic ulcers in animals with biliary fistulas, and have referred to the recent literature on the formation of ulcer following exclusion of bile from the intestine.

METHOD OF EXPERIMENTATION

Under ether anesthesia and with aseptic technic, obstructive jaundice was produced by double ligation and section of the common bile duct. The gallbladder was also removed from most of the animals, and from one to three lobes of the liver were removed at the same time from a small number. The animals were then maintained on a diet of dog biscuits, milk and corn syrup, with an occasional feeding of meat in a few instances; a few animals were given a coarser diet consisting of a cooked mixture of ground corn meal, ground bone and meat scraps. The latter diet was instituted to determine the effect of the rough element in the food on the production of ulcer.

Frequent examinations were made of the feces of these animals, and the absence of bile from the gastro-intestinal tract was verified by tests for urobilin. Only occasionally an animal showed evidence of reestablishment of biliary drainage into the intestine, and in such animals it was clearly manifest by the normal color of the stools, urobilin in the feces and the complete disappearance of the serum bilirubinemia.

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* Submitted for publication, March 9, 1931.

* From the Division of Experimental Surgery and Pathology, the Mayo Foundation.

1. Berg, B. N., and Jobling, J. W.: Biliary and Hepatic Factors in Peptic Ulcers: An Experimental Study, Arch. Surg. **20**:997 (June) 1930.

RESULTS OF EXPERIMENTS

Necropsy of sixty-four animals from five to two hundred and ninety-five days following ligation of the common bile duct showed acute, subacute or chronic peptic ulcer of the duodenum or stomach. During the same period only twenty-three animals died of causes attributable to this operation without showing gross evidence of peptic ulcer; their deaths occurred from twenty-one to one hundred and ninety-five days following ligation of the common bile duct. These data are summarized in tables 1 and 2.

The gross appearance of the ulcers were varied; some appeared as acute perforations with little, if any, reaction at the margins of the perforation. The other extreme was characterized by the typical crater ulcer with overhanging edges and indurated base. In general, the ulcers that seemed most acute occurred in the early months of jaundice, but in some cases moderately indurated ulcers of the subacute type were found after from three to six weeks of obstructive jaundice. Again, acute perforating ulcers have been noted after eight months of jaundice, and in some cases acute perforations were found associated with another ulcer that appeared quite chronic, and in one specimen, an acute erosion had passed through a portion of a definite subacute gastric ulcer.

Of the sixty-four animals with peptic ulcer, eight had gastric ulcers, which, with one exception, were on the pylorus and were almost all bordering on the line of the lesser curvature of the stomach. One perforating ulcer was situated at the incisura on the line of the lesser curvature. In five cases both gastric and duodenal ulcers were present. Sixty-one animals had duodenal ulcers, thirty-eight of which appeared to be single; twenty-three animals had from two to four ulcers. The duodenal ulcers were situated about 1 cm. beyond the pylorus, slightly anterior to the line of the lesser curvature of the stomach. Some ulcers showed a definite tendency to encircle the duodenum a short distance beyond the pylorus. If a second duodenal ulcer was present, it was about 1 cm. below the first ulcer and approximately on the opposite side of the duodenum. A third ulcer, if present, was below the second and again on the opposite side of the duodenum.

That these ulcers are not the result of trauma at the time of operation is evidenced by the fact that many of the animals did not show evidence of ulcer for many months after operation. Also, in a large number of animals denervation of the liver was accomplished by stripping the adventitia from the hepatic artery, portal vein and common bile duct, with section of all the other structures of the gastrohepatic omentum. Many of these animals have survived for more than two years, and peptic ulcers have not been observed in any case.

TABLE 1.—*Peptic Ulcer in Dogs Following Ligation of the Common Bile Duct*

Dog	Weight, Kg.	After Ligation, Days	Ulcers Present	Probable Cause of Death
1	11.5	5	1 duodenal	Peritonitis
2	7.8	6	2 duodenal	Distemper
3	9.4	8	1 duodenal	Perforation
4	6.0	8	3 duodenal	Perforation
5	20.0	9	4 large duodenal	Perforation
6	6.8	10	1 duodenal	Perforation
7	7.3	13	1 duodenal	Perforation
8	15.1	14	2 duodenal	Perforation
9	10.2	15	2 duodenal	25 per cent of liver also removed; peritonitis
10	8.8	15	2 duodenal	25 per cent of liver also removed; perforation
11	13.4	17	2 duodenal	Perforation
12	5.9	21	1 duodenal	Perforation
13	6.4	23	1 duodenal	Intestinal hemorrhage
14	7.4	24	2 duodenal	Rupture of biliary duct
15	7.2	25	2 duodenal	Perforation
16	13.0	27	2 duodenal	Anemia
17	7.6	28	3 duodenal	Perforation
18	9.2	28	1 duodenal	Rupture of biliary duct
19	8.5	29	1 duodenal; 1 pyloric	Intestinal hemorrhage
20	6.0	30	1 duodenal	Perforation
21	10.1	31	1 healing duodenal	Intestinal hemorrhage
22	9.2	35	1 duodenal	30 per cent of liver also removed; perforation
23	5.2	35	1 duodenal	Intestinal hemorrhage
24	14.5	36	1 duodenal	Intestinal hemorrhage
25	14.4	39	1 duodenal	Inanition
26	15.1	39	1 duodenal	Perforation
27	12.6	41	2 duodenal	Perforation
28	6.0	41	1 pyloric	Hemorrhage from ulcer
29	6.8	42	1 healing duodenal	Intestinal hemorrhage
30	8.2	43	1 duodenal	Intestinal hemorrhage
31	19.8	47	2 duodenal	Perforation
32	7.5	55	1 duodenal	Perforation
33	10.9	59	1 duodenal	Subdiaphragmatic abscess
34	17.3	60	1 duodenal	Perforation
35	13.5	60	1 duodenal	Hemorrhage from ulcer
36	6.7	63	2 duodenal	30 per cent of liver also removed; perforation onto liver
37	18.2	66	1 duodenal	Empyema
38	6.2	68	1 healing duodenal	35 per cent of liver also removed; inanition
39	10.5	70	2 duodenal; 1 pyloric	Intestinal hemorrhage
40	9.9	72	1 duodenal	Hemorrhage from ulcer
41	9.3	76	1 duodenal	Perforation
42	24.0	78	2 duodenal	50 per cent of liver also removed; anemia
43	7.6	81	2 duodenal	Perforation
44	7.2	81	1 duodenal	35 per cent of liver also removed; subdiaphragmatic abscess
45	6.5	84	2 duodenal	60 per cent of liver also removed; perforation
46	15.4	85	1 healing duodenal	Hemorrhage from ulcer
47	5.4	85	1 pyloric	Inanition
48	10.7	87	1 duodenal	Perforation
49	11.8	88	1 duodenal	Perforation of ulcer into gallbladder
50	4.7	93	1 duodenal	Perforation
51	9.9	98	1 duodenal	Hemorrhage in stomach
52	3.4	101	1 duodenal	Inanition
53	15.3	112	1 healing duodenal	Cholangeitis
54	6.5	118	2 duodenal; 1 gastric	Perforation
55	17.0	121	1 duodenal	35 per cent of liver also removed; intestinal hemorrhage
56	9.2	128	1 duodenal	35 per cent of liver also removed; intestinal hemorrhage
57	13.5	136	2 duodenal; 1 gastric	Perforation
58	6.0	159	1 pyloric	25 per cent of liver also removed; inanition
59	6.7	168	2 duodenal	Hemorrhage from ulcer; 35 per cent of liver also removed
60	6.5	174	1 duodenal; 1 pyloric	Inanition
61	13.5	176	1 duodenal	50 per cent of liver also removed; anemia
62	5.5	179	1 duodenal	30 per cent of liver also removed; anemia
63	20.6	235	1 duodenal	Infection of biliary tract
64	8.2	295	1 duodenal	35 per cent of liver also removed; intestinal hemorrhage

COMMENT

We have eliminated from consideration here a large number of animals in which the common bile duct had been ligated. In a very few animals, biliary connection was reestablished to the gastro-intestinal tract and relief from jaundice was afforded. Many others that had been jaundiced for several months died as the direct result of other experiments. Only a small percentage of these animals had peptic ulcer, but since their death was due to causes other than sequelae of the ligation of the common bile duct, this entire group was not considered. From the data presented it is evident that the occurrence of peptic ulcer depends on factors not directly related to the duration of jaundice. From

TABLE 2.—*Dogs Without Gross Peptic Ulcer Following Ligation of the Common Bile Duct*

Dog	Weight, Kg.	After Ligation, Days	Probable Cause of Death
1	10.2	22	Intestinal hemorrhage and anemia
2	9.2	22	Perforation of biliary duct
3	4.9	27	Gastric and duodenal hemorrhage
4	8.9	29	Gastric hemorrhage
5	9.4	31	Perforation of biliary duct
6	6.1	40	Inanition
7	6.0	40	25 per cent of liver also removed; intestinal hemorrhage
8	5.4	43	Inanition
9	7.1	49	Perforation of biliary duct
10	6.9	51	Intestinal hemorrhage
11	7.8	68	Intestinal hemorrhage
12	10.9	71	Cholangeitis
13	11.3	80	Inanition
14	9.0	89	30 per cent of liver removed; inanition
15	15.5	93	Pulmonary edema; ascites
16	10.8	95	40 per cent of liver also removed; inanition
17	10.7	114	50 per cent of liver also removed; inanition
18	5.1	119	Intestinal hemorrhage
19	10.7	132	Inanition
20	6.4	133	Bronchopneumonia
21	7.8	141	25 per cent of liver also removed; inanition
22	5.0	142	Inanition
23	5.2	195	Intestinal hemorrhage

the data that we have not included we could add further evidence similar to that in our group of jaundiced animals that died without the development of ulcer. These animals demonstrate that with complete obstruction of biliary outflow, life may continue for several months without the formation of ulcer. Several of our jaundiced animals have died as the result of other experiments after eleven months of obstructive jaundice, and few in this particular group showed evidence of peptic ulcer.

The most common cause of death in these jaundiced dogs was perforation of a peptic ulcer with resultant peritonitis. The next most common cause of death was hemorrhage into the gastro-intestinal tract; for the most part this was evidenced by chronic oozing with fresh blood in the feces or tarry stools. In some instances there were several periods of intestinal bleeding, with remissions and subsequent improvement in

the general condition of the animal. Transfusion was resorted to in many instances, after which blood usually continued to appear in the stools for two or three days and then was absent for a few weeks. Intramuscular injection of from 5 to 10 cc. of whole blood also seemed to be effective in bringing about remission, but several animals continued to bleed until death. In some animals massive hemorrhage occurred from the site of the peptic ulcer.

Definitely diminished resistance to the formation of ulcer appears to be present following ligation of the common bile duct. This may be due at least in part to the exclusion of bile from the gastro-intestinal tract. Kapsinow [2] (1926) found that in seventeen of forty-three dogs, duodenal ulcer developed in about two weeks following anastomosis of the gallbladder to the renal pelvis and ligation of the common bile duct. We have not been successful in preventing the formation of ulcer in a number of dogs by oral administration of gallbladder bile twice daily. It should be noted, however, that such administration of bile differs greatly from the normal flow into the intestine, and that it was administered to dogs that already had complete biliary retention.

Dietary factors play a definite part in the formation of peptic ulcer in dogs with obstructive jaundice. For the most part, in our animals maintained on a bland diet of milk, syrup and bread, ulcer did not develop nearly as soon as in others maintained on a coarser diet of cooked corn meal, meat and ground bone. We are inclined to attribute this difference to the mechanical factors of the formation of ulcer, although chemical factors are probably also concerned. One striking observation should be mentioned in this respect: most of the animals that died of acute perforation of an ulcer were those that maintained an excellent appetite and always ate their food rapidly; thus ulcers were found in well nourished animals up to a few days before death. On the other hand, many of the animals lost appetite and weight, and died without the formation of ulcer. With the exceptions mentioned, all of our animals were given the same food, and all were kept under the same hygienic conditions for the entire period. However, there was a definite tendency for ulcers to form in groups of animals; that is, the lapse of several weeks without the death of an animal from ulcer would be followed by several deaths in the same week without reference to the duration of jaundice. We have been unable to ascribe this periodicity to diet, hygienic conditions or other known factors.

We do not feel justified in attempting to interpret our observations of jaundiced dogs in relation to the occurrence of peptic ulcer in man. It is obvious that diminished resistance to the formation of ulcer is pri-

2. Kapsinow, Robert: The Experimental Production of Duodenal Ulcer by Exclusion of Bile from the Intestine, Ann. Surg. **83**:614 (May) 1926.

Fig. 1.—Pylorus and duodenum opened along the line of the greater curvature of the stomach, nine days after ligation of the common bile duct. The animal died of peritonitis subsequent to the perforation of the ulcer.

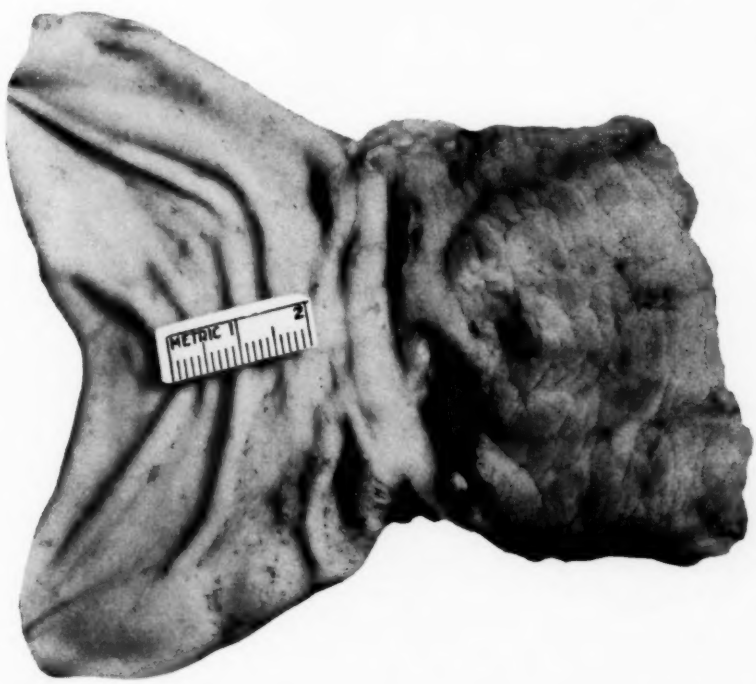

Fig. 2.—Pylorus and duodenum seventy-eight days after ligation of the common bile duct and removal of approximately 50 per cent of the liver. Severe anemia contributed to the cause of death in this animal.

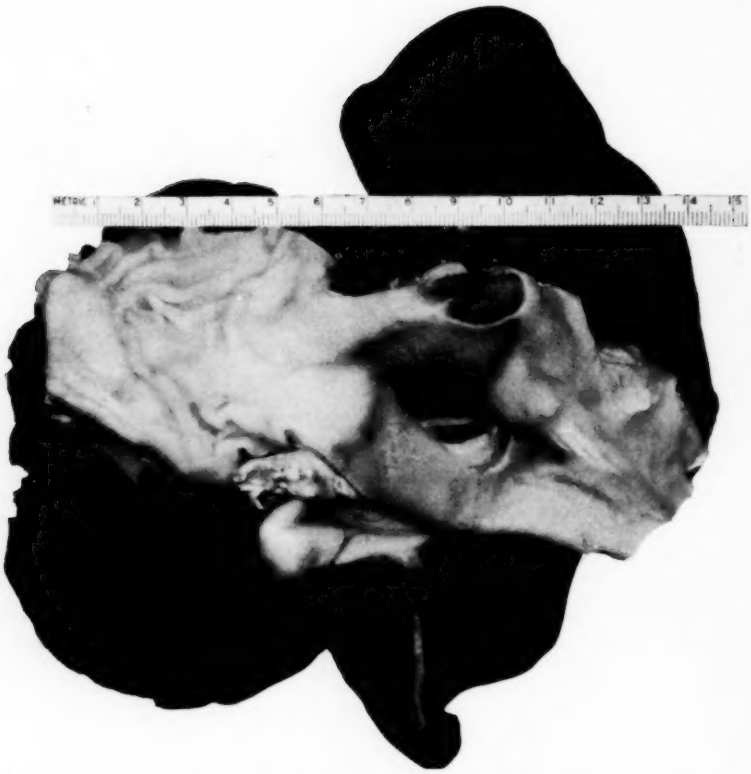

Fig. 3.—Pylorus and duodenum on the liver of a dog eighty-seven days after ligation of the common bile duct. Two duodenal ulcers were present, one of which has perforated into the gallbladder. The animal died of inanition.

Fig. 4.—Stomach and duodenum from a dog one hundred and eighteen days after ligation of the common bile duct. Acute perforation developed on the base of an apparently older gastric ulcer.

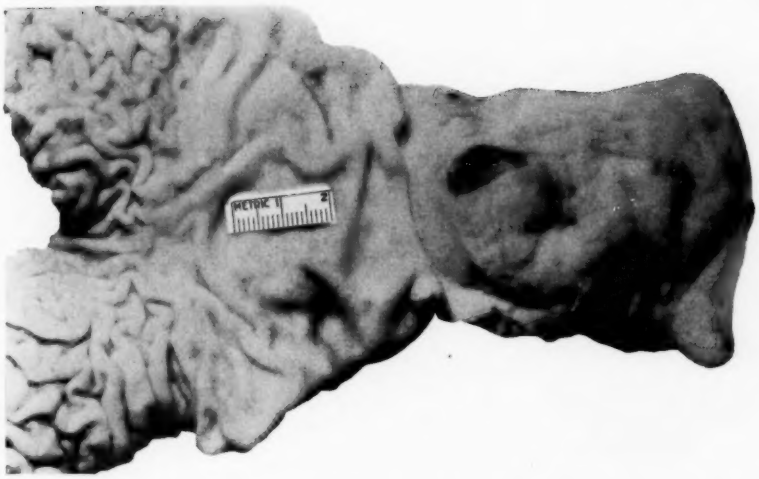

Fig. 5.—Stomach and duodenum one hundred and twenty-one days after ligation of the common bile duct and removal of approximately 35 per cent of the liver. The animal was killed at this time so that specimens of tissues could be obtained.

Fig. 6.—Stomach, duodenum and liver one hundred and sixty-eight days after ligation of the common bile duct and removal of 35 per cent of the liver. The animal died following a massive hemorrhage from the base of the large duodenal ulcer which had also eroded into the liver.

Fig. 7.—Pylorus and duodenum showing a large chronic duodenal ulcer two hundred and thirty-five days after ligation of the common bile duct. Marked cholangeitis was also present.

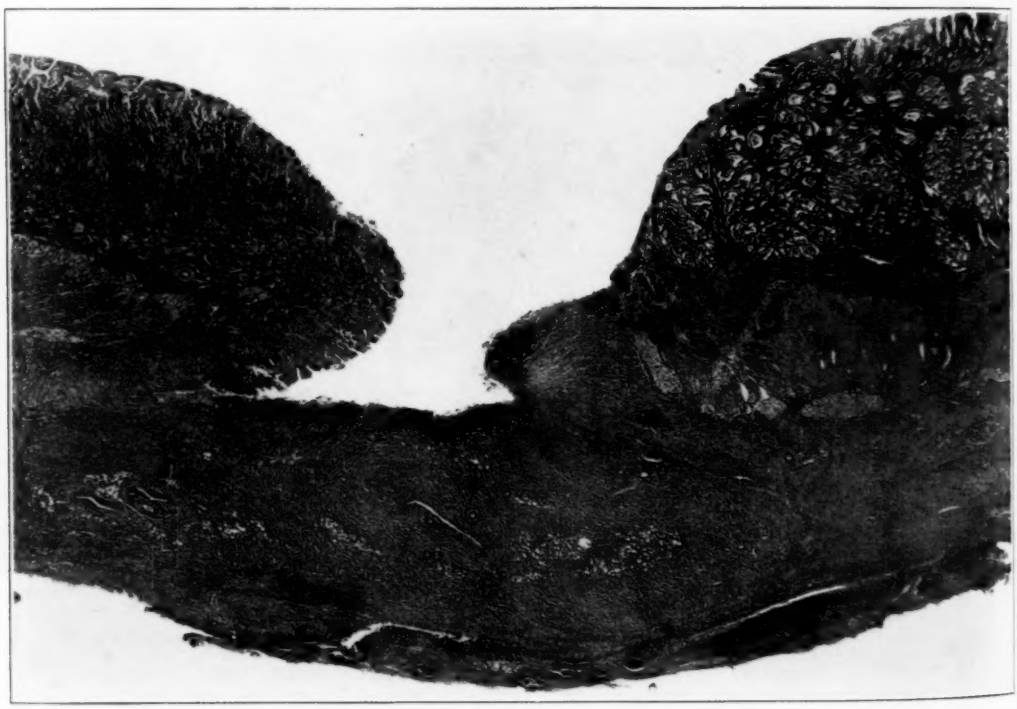

Fig. 8.—Section of duodenal ulcer occurring in a dog ninety-three days after ligation of the common bile duct; reduced from a magnification of × 10.

marily responsible for both, but the specific cause has not been demonstrated. With reference to obstructive jaundice in man, it should be noted that few human beings have complete biliary obstruction for more than a few months, and we have had a number of dogs with complete obstruction for almost a year without the development of peptic ulcer. If the difference in the vital span of these two species is considered, unrelieved jaundice would have to be present for a long time in the human being to be comparable with that in the animals under consideration here (figs. 1 to 8).

SUMMARY

Following ligation of the common bile duct there is marked decrease in resistance to the formation of peptic ulcer in dogs. In sixty-four animals, acute, subacute or chronic duodenal or gastric ulcers developed in from five to two hundred and ninety-five days after operation. Only twenty-three animals with uncomplicated complete obstructive jaundice, living from twenty-two to one hundred and ninety-five days, died of causes referable to ligation of the common bile duct without showing gross evidence of peptic ulcer.

TRAUMATIC NECROSIS OF THE LIVER WITH EXTENSIVE RETENTION OF CREATININE AND HIGH GRADE NEPHROSIS *

FERDINAND C. HELWIG, M.D.
AND
THOMAS GROVER ORR, M.D.
KANSAS CITY, MO.

The possible relationship between the concurrence of a liver severely damaged by trauma and a high grade nephrosis with extreme retention of creatinine leads us to a consideration of the mechanism by which such a picture might be produced. We could find a record of but one case that in any manner paralleled the one reported here.

REPORT OF A CASE

History.—H. C., a white boy, aged 15, was admitted to St. Luke's Hospital on June 20, 1930, and died on July 1, 1930. He was injured when an automobile turned over and a portion of the car rested on his chest and abdomen.

When he entered the hospital, he was in a state of shock. Examination revealed an abrasion on the left thoracic wall, fracture of the right clavicle, subcutaneous emphysema above and below the right clavicle and tenderness along the right costal margin and in the region of the right kidney. No abdominal rigidity was present. The temperature was 96 F. and the pulse rate, 120. On the day of admission the temperature rose to 102.8 F. and returned to normal within two days. The day before death it again rose to 101 F. Roentgen examination revealed fractures of the right clavicle and the right fifth, sixth and seventh ribs. A catheterized specimen of urine showed a few red blood cells and albumin. This finding continued throughout the illness. The patient vomited whenever water was taken by mouth. On the second day after admission he complained of abdominal soreness, and the tenderness along the right costal margin and in the region of the kidney increased; definite jaundice also appeared. On the fifth day fluid could be demonstrated in the abdominal cavity. It was thought that the gallbladder probably was ruptured.

On the sixth day, an exploratory laporotomy was done. An incision was made in the upper right rectus muscle with the patient under local anesthesia. The gallbladder was found intact. The abdominal cavity was filled with bloody, bile-stained liquid. Drainage was instituted, and the wound was closed without further exploration. The general condition of the patient was critical.

When the patient was admitted to the hospital, the blood pressure was subnormal. During the illness it gradually rose until it reached 150 systolic and 80 diastolic before death. The blood count showed secondary anemia and slight leukocytosis. During the first six days of the illness, the patient was given large quantities of 10 per cent dextrose intravenously and physiologic solution of sodium chloride. The latter, however, was reduced because of the suppression of urine. At no time during the illness did the patient pass more than 200 cc. of urine a day. Generalized edema developed. Vomiting continued daily during the entire

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* From the Departments of Surgery and Pathology, St. Luke's Hospital.

illness. The jaundice, which developed on the second day, continued until death. The patient died on the eleventh day following injury. He was conscious until the last two days.

The results of the chemical examination of the blood and urine are recorded in tables 1 and 2.

Necropsy.—The body was that of a large, well nourished boy. It measured 6 feet (182.9 cm.) in length and weighed 185 pounds (83.9 Kg.). The most striking feature on external inspection was the extreme degree of jaundice affecting the skin, sclera and mucous membranes. A low grade generalized edema was also present. A superficial cutaneous abrasion was seen over the fractured right clavicle. The latter crepitated readily on manipulation and when, on exploration, we found the fracture surrounded by a wide zone of both old and fresh hemorrhage. An abdominal incision, partly healed and sutured, was present in the

TABLE 1.—*Chemical Analysis of the Blood*

Date	Nonprotein Nitrogen, Mg. per 100 Cc.	Creatinine, Mg. per 100 Cc.	Sugar, Mg. per 100 Cc.	Whole Blood Chlorides, Mg. per 100 Cc.
6/22/30	75	3.7	106	440
6/23/30	120	6.0	114	495
6/24/30	150	13.0	101	500
6/25/30	150	14.0	...	...
6/26/30	146	18.0	...	...
6/28/30	200	22.0	121	515
6/29/30*	240	25.0	...	...
6/30/30	240	25.0	...	500

* The carbon dioxide combining-power of the blood plasma was 24 per cent by volume.

TABLE 2.—*Chemical Analysis of the Urine*

Date	Total Nitrogen, Gm. per 100 Cc.	Urea, Gm. per 100 Cc.	Urea Nitrogen, Gm. per 100 Cc.	Chlorides, Gm. per 100 Cc.	Total Urine in 24 Hours, Cc.
6/27/30	0.613	2.88	.....	0.575	125
6/28/30	0.583	0.988	0.462	0.44	110
6/29/30	0.491	1.44	.....	0.57	175
6/30/30	0.8	1.6	0.7	0.736	160

region of the right rectus muscle in the upper part of the abdomen. This incision held three rubber drains.

The peritoneal cavity contained about 2 liters of bright red, very bloody fluid without clots. The liver was somewhat enlarged, weighing 2,330 Gm. and measuring 29 by 20 by 10 cm. It was greenish brown. On the upper surface of the right lobe immediately under the diaphragm, there was a laceration of Glisson's capsule about 5 cm. in length, which was spread apart about 2 cm., revealing the substance of the liver beneath. In the midline, between the right and left lobes on the anterior surface, there was a discolored, reddish mottled, elongated area, which was slightly depressed. This irregular band of discoloration varied from 2 to 4.5 cm. in diameter and extended along almost the entire anterior superior exposed surface out to the free margin. The hepatic tissue beneath this band was softer in consistency than that adjacent to it, and when sectioned it was soft, hemorrhagic and pulpified. The capsule over it was not ruptured.

On cross-section, this pulpified hepatic tissue was found to extend over an area roughly approximating the size of the clenched fist. It was so located as to include the larger proximal ramifications of the right and left hepatic ducts. In the tissue

not involved in the pulpification, the bile ducts were considerably dilated, and the parenchyma of the liver was pigmented with bile. When the extrahepatic ducts were traced into the liver, we found the traumatized area to extend within about 2 cm. of their entrance into the substance of the liver.

An incidental finding was a cavernous hemangioma about 4 cm. in diameter extending into the liver like a wedge on the free edge anteriorly, about 5 cm. lateral to the midline and entirely outside the traumatized zone.

The gallbladder was somewhat distended with thick, black, viscid bile which could be forced into the duodenum through the papilla of Vater only when great pressure was exerted. The mucosa of the duodenum was edematous and deep yellow. The remainder of the small intestine showed nothing but a few subserous petechiae. In the large intestine both submucous and subserous hemorrhages were diffusely distributed, some of them measuring as much as 1 cm. in diameter.

The kidneys were greatly swollen and softened, and of about equal size. They weighed together 580 Gm. When sectioned, the capsules retracted and stripped easily, revealing a dark greenish-red, mottled, cortical surface. The outlines between the medulla and cortex were distinct, owing to a rather intense hyperemia of the pyramids. The cortex had a swollen lusterless appearance suggesting a high grade degenerative change. The blood vessels showed no emboli or thrombi, and the ureters and bladder were essentially normal.

Both pleural cavities contained about 250 cc. of bright red, unclotted, very bloody fluid, and the lungs were partially collapsed. In both lungs interstitial, parenchymatous and subpleural hemorrhages were seen, some measuring as much as 15 mm. in diameter. No areas of pneumonia were found. The right fifth, sixth and seventh ribs were fractured, but had not penetrated the parietal pleura.

Aside from the extreme pigmentation of the bile of all the viscera, no other noteworthy findings were encountered.

Histologic Pathology.—Heart: The myocardial fibers showed some swelling and considerable fragmentation and segmentation. The epicardial mesothelial cells were almost cuboidal and showed some proliferation. There was some accumulation of green pigment in most of the cells.

Lungs: Many deeply pigmented cells were found in the alveoli which were probably exfoliated, alveolar epithelial cells. Masses of red cells, sometimes intermingled with stringy, fibrinous material, were seen in many of the alveoli, but no exudate of pus cells was present.

Liver: Glisson's capsule was intact. It showed some pigmentation with bile. The lobulation of the nontraumatized area was fairly distinct. About the central veins the hepatic cells showed some shrinkage and cytoplasmic granulation. Between many of the cells in this region of the lobule the biliary canaliculi and capillaries were plugged with bright green pigmented material which was not actually seen in the liver cells but was found at the points where the fine capillary twiglets entered, and where they ran between the cells. The stellate cells of Kupffer were swollen, and many of them contained some greenish, granular pigment. Many were partially torn away from the columns of hepatic cells and seemed to be desquamated into the sinusoids.

In the pulpified region, we saw large areas of hemorrhage in which no viable hepatic cells were present. Dead, non-nucleated, amorphous shadows, staining faintly eosinophilic and presenting the typical outline of parenchymatous cells of the liver, were found in masses scattered about in these hemorrhagic areas. In some respects these zones resembled those seen in eclampsia. The areas were surrounded by a thick zone of fibroblastic appearing cells in which proliferating bile ducts were numerous, and occasional disintegrating hepatic cells were seen. The fibroblastic cells were growing into these areas of hemorrhage. Peripheral

to these fibroblastic zones, the parenchyma of the liver resembled the remainder of the liver previously described.

Spleen: The capsule showed some greenish pigmentation, and considerable green granular pigment was scattered about in the pulp. Red cells were only infrequently encountered here, and then they appeared as mere faintly pigmented, pale shadows. Scattered eosinophils were seen in the pulp. The malpighian bodies were not distinctly outlined.

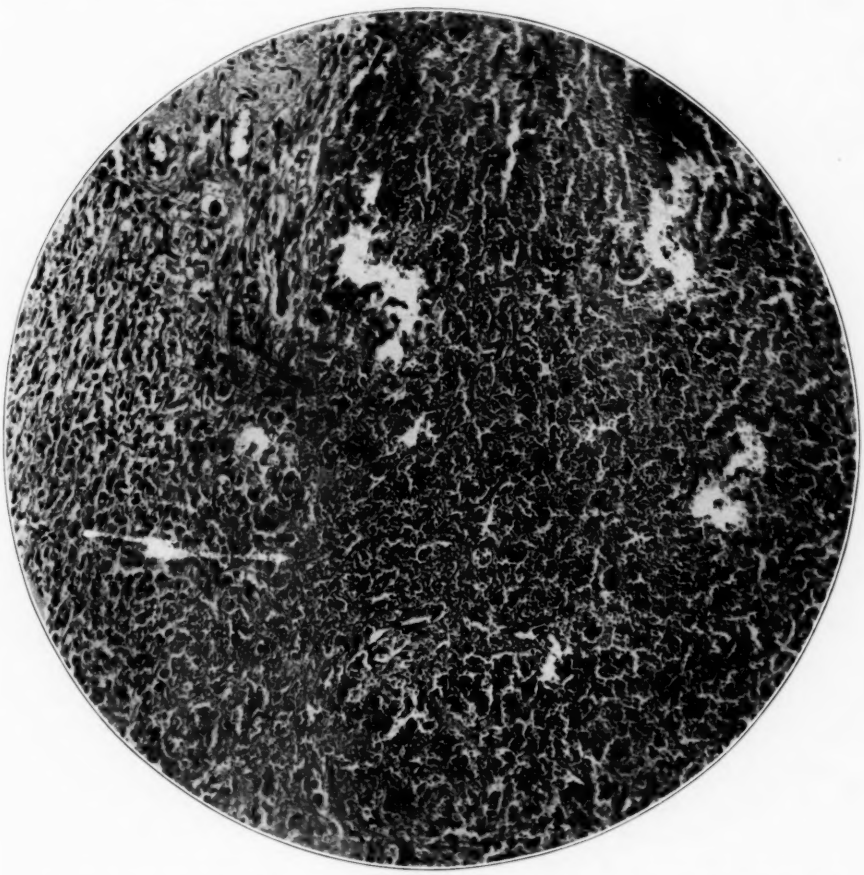

Fig. 1.—Low power photomicrograph of the liver showing the border of the area of hemorrhagic, necrotic pulpification. The upper left portion shows the fibroblastic zone with organization; the extreme right side, necrotic hepatic cells; the center, predominant hemorrhage; × 125.

Pancreas: The pancreas was histologically normal.

Gastro-Intestinal Tract: The duodenum in the region of the papilla of Vater showed a swollen mucosa and submucosa. The epithelium lining the villi was almost entirely exfoliated, and the submucosa looked dead. It was anuclear and pigmented a diffuse greenish hue. The hemorrhagic areas in the small and

large intestine showed desquamation and granular disintegration of the epithelium, and in the epithelial stroma rather extensive focal hemorrhages were present.

Kidneys: In the medullary portion scattered foci of leukocytes were seen. Monocytes, plasma cells and eosinophils were present in large numbers. Here the substance of the kidney seemed to be completely disorganized, and some fibroblasts were seen in these irregular focal leukocytic accumulations. Some scattered interstitial hemorrhages were also encountered, and many of the small ves-

Fig. 2.—Low power photomicrograph of the kidney showing vascular congestion, hemorrhage and interstitial leukocytic reaction secondary to degenerative changes; × 125.

sels and capillaries were widely dilated with red cells; some of the latter appeared to be undergoing hemolysis. Many of the tubuli of the medulla also contained red cells. In addition to cloudy swelling, a granular, albuminous precipitate and pale casts were found in the collecting tubules. Some of the latter showed regeneration of the epithelium. In the cortex, the convoluted tubules and loops of Henle showed an extensive parenchymatous degeneration. Many tubules were seen in which the epithelial cells contained only two or possibly three nuclei; these

nuclei were pale and hazy and apparently undergoing karyolysis. Some tubules contained no nuclei and seemed to have undergone complete necrosis. The cytoplasm of the epithelium of the more highly differentiated tubules was generally pigmented with greenish, fine, dusty granules. The glomeruli showed swelling of the epithelium lining Bowman's capsule, and there were red cells scattered about in the space between the tuft and the capsule. In the capillary loops the endothelium was somewhat swollen and a few polymorphonuclear leukocytes were seen here and there in the tortuous capillary channels.

Special fat stains used on sections from both kidneys showed no evidence of fatty degeneration or fat embolism.

Fig. 3.—Oil immersion photomicrograph showing cortical renal tubule with absence of nuclei, nuclear shadows, karyolysis of the remaining swollen nuclei and stringy precipitate of albumin in the tubular lumen; × 500.

COMMENT

In 1927, Furtwaengler,[1] from Professor Clairmont's clinic in Zurich, reported the case of a woman, 22 years old, who suffered a crushing injury to the liver in an automobile accident. She lived only three days, and in this period presented a clinical course almost identical with that of our patient, with the exception that she was not jaundiced. At necropsy a bilateral necrosis of the cortices of both kidneys was found.

1. Furtwaengler, A.: Diffuse Rindennekrose beider Nieren nach Leberruptur, Krankheitsforschung **4**:349, 1927.

Furtwaengler believed that this patient had a latent tendency to vascular spasm due to some nervous disposition and that the cortical renal necrosis resulted from an ischemia produced by a hypothetic chemical toxin circulating in the blood, which toxin arose from decomposition of hepatic tissue. The toxin thus caused the vascular spasm which was followed by ischemia and subsequent renal necrosis. Among other possibilities that he ruled out were fat embolism and bilateral thrombosis or embolism of the renal veins or arteries.

Our case differs from Furtwaengler's only in degree. Careful histologic examination of the kidney showed both inflammatory and degenerative changes. This degeneration had proceeded to actual tubular epithelial necrosis in many of the more highly differentiated tubules, but was not so advanced as that described by Furtwaengler.

A certain degree of renal change might be explained on the basis of extensive jaundice with the bile acting as a poison to the renal epithelium. Dead hepatic tissue might also have produced some toxin which, when absorbed, acted on the kidney. In our case the jaundice was not wholly obstructive, since the stools remained colored up to the time when the patient began to bleed from the serous and mucous surfaces. There was, however, in the liver some degree of biliary obstruction from pressure of the pulpified hepatic tissue and hemorrhage on the larger hepatic radicles. Histologic examination of the liver showed a considerable degree of plugging of the biliary canaliculi. The marked degree of jaundice in our case and the possible decrease in the ability of the liver to excrete fibrinogen might account for the extensive bleeding in the latter period of the patient's illness. Moreover, some of the jaundice could possibly have resulted from the severe bleeding with absorption of hemoglobin derivatives.

The extreme retention of creatinine was distinctly out of proportion to the retention of other nitrogenous products. Although the terminal figures for nonprotein nitrogen were high, these values were in no way comparable to the remarkable rise of creatinine in the blood to 25 mg. per hundred cubic centimeters. The marked decrease in nitrogen in the urine was striking, although it was about what might be expected in a person in whom the retention of nitrogen in the blood was so high and the urinary output so scanty.

There is yet another action that may arise from biliary obstruction which could have been present in our case, namely, the elaboration of an active vasodepressor substance in the liver. After tying the common bile ducts of dogs, Radvin [2] obtained from their livers an extract containing both histamine and choline which was a potent vasodepressor.

2. Ravdin, I. S.: Vasodepressor Substances in the Liver After Obstruction of the Common Duct, Arch. Surg. **18**:2191 (May) 1929.

Recently McEnery, Myer and Ivy,[3] through the production of mechanical ischemia of the kidney, were able, in a short period, to cause a condition in dogs clinically simulating uremia, from which the animals died. Scriver and Oertel [4] in a discussion of necrotic sequestration of the kidney in pregnancy, explained their findings on the basis of a vasoparalysis with subsequent thrombosis. Beneke and Schmorl [5] on the other hand, felt that the necrotic changes seen in the so-called "pregnancy kidney" of eclampsia were due to some pressor substance elaborated by the liver and producing vascular spasm, ischemia and subsequent necrosis. Moreover, such observers as Griesinger and Leyden [5] held that the necrosis seen in the kidney in cholera was due to vascular spasm and ischemia. However, Fraenkel [5] explained this necrosis of the kidney as being due to the direct action of the cholera toxin. Scriver and Oertel [4] believed that the idea of vascular spasm and ischemia, as well as "the rather hazy theories of primary toxic cell degeneration," does not explain the changes in the kidney as logically as their own hypothesis of vasoparalysis.

Just what the relationship between the kidney and the liver may be is not definitely understood. The apparent adverse effects of disease of the hepatic and biliary tract on the kidney have been noted by such authors as Fitz-Hugh [6] and Zaffagnini,[7] and the harmful effect on the kidney seen in some cases of obstruction of the common bile duct is a well recognized fact. We have seen a patient with a previously undamaged kidney who died from uremia with extensive intestinal hemorrhage several days after a cholecystectomy was performed for removal of a stone. The liver showed a moderately severe hepatitis, and the renal changes were remarkably similar to those in the case presented here. Walters and Parham,[8] in a case of obstruction of the common bile duct without operation, saw extensive intestinal hemorrhage and death from uremia. The exact mechanism of this renal damage is not clearly understood.

In our case several possibilities are present that might be considered of etiologic importance; all of them may have been of some impor-

3. McEnery, E. T.; Myer, J., and Ivy, A. C.: Studies on Nephritis, J. Lab. & Clin. Med. **12**:349, 1927.

4. Scriver, W. de M., and Oertel, H.: Necrotic Sequestration of the Kidneys in Pregnancy, J. Path. & Bact. **33**:1071, 1930.

5. Beneke, Schmorl, Griesinger, Leyden, Fraenkel, Gerhardt and Ogata, quoted by Kaufmann: Pathology for Students and Practitioners, authorized translation, Stanley P. Reimann, Philadelphia, P. Blakiston & Son, 1929.

6. Fitz-Hugh, Thomas, Jr.: Hepato-Urologic Syndromes, M. Clin. North America **12**:1101, 1929.

7. Zaffagnini, A.: Hematuria and Gallstones, Arch. ital. di urol. **4**:161 (Dec.) 1927.

8. Walters, W., and Parham, D.: Renal and Hepatic Insufficiency in Obstructive Jaundice, Surg., Gynec. & Obst. **35**:605, 1922.

tance in producing the complete pathologic picture. We must consider the pulpified, necrotic liver as possibly elaborating some toxin which, through the blood, acted on the kidney or its blood supply. We must also consider the jaundice that appeared early in the disease and increased progressively until death. And we must not disregard traumatic shock and trauma to the kidney itself. Regarding the last factor, it might be mentioned that no evidence of trauma was found at necropsy, although the patient had blood in the urine at the first examination. Trauma alone has been considered by Neubürger [9] to be the cause of an angiospasm of the renal artery of sufficient intensity to cause ischemic infarction without thrombosis in several cases that he has investigated.

As regards jaundice, the picture produced in our case was not that usually found in necrosis caused by bile. Although the kidney was pigmented, the typical "grass-green kidney" was not seen. However, we do know that bile acting on the protoplasm of the more highly differentiated epithelium of the kidney, particularly the convoluted tubuli, leads at times to actual epithelial necrosis (Gerhardt and Ogata [5]).

The multiple factors in our case somewhat clouded the development of the picture. However, we feel that, in the absence of thrombosis, neither vasoparalysis nor vasoconstriction alone could have caused the renal picture. It seems more logical to assume that some potent toxin was produced in the liver from hemorrhagic necrosis which acted directly on the parenchymatous cells of the kidney, since the histologic picture produced was that of a slow degenerative change proceeding to necrosis with the superimposed inflammatory reaction secondary to the degenerative change. Many of the tubules in the kidney showed only cloudy swelling, while others had lost their nuclei by karyolysis, and not infrequently nuclei were seen in an incomplete stage of autolysis.

CONCLUSIONS

A case of traumatic pulpification of the liver without rupture is reported in which the patient died after eleven days, with an extensive nephrosis. He was extremely jaundiced and had diffuse hemorrhages into the serous cavities. The output of urine was scanty, and the blood nitrogen was greatly increased, particularly the creatinine, which amounted to 25 mg. per hundred cubic centimeters. The mechanism of renal necrosis is discussed, and the histologic renal picture, as well as the clinical course, would seem to favor the theory that some toxin was elaborated in the liver through hemorrhagic necrosis which acted directly on the kidney.

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9. Neubürger, K.: Angiospasm as a Cause of Renal Infarcts, Virchows Arch. f. path. Anat. **265**:789, 1927.

CANCER AND WEIGHT *

CHARLES C. LUND, M.D.
BOSTON

The influence of diet on cancer has been intensively studied for the last twenty years by means of experimentation on animals. As a result of this study, it may be stated that either certain vitamin deficiencies or low caloric diets will delay or prevent the taking of transplants of nearly all the transplantable tumors. On the other hand, dieting after the implant is established has no effect on many tumors, and a comparatively insignificant one on others. Tumors may stop growing when abnormal diets are used, but the animals seldom survive any longer than those on a normal diet and are never cured by such means. Much of the recent work in this field is referred to in an article by Henry Jackson, Jr.[1]

In the clinics, little attention has been paid to diet in cases of cancer, and no well controlled experiments have been recorded in the literature that indicate any worth while results from dietary treatment. For centuries, there have been sporadic developments of starvation or other dietary treatment, but none of them have lasted long or been taken up widely.

If diet does not influence the cure of cancer, there are, however, some data at hand that indicate a possible relationship between bodily nutrition and the etiology of cancer. In Hoffman's[2] book may be found some figures that show that people who are overweight when they take out life insurance are more liable to die from cancer than people who are of normal weight. People who are underweight have a reduced liability. Further work on this subject has been done by Dublin and Jackson and is published in Jackson's[3] paper. Their figures show an increased liability to death from cancer of nearly 50 per cent in obese persons as compared with those who are underweight. It is interesting that figures compiled from such data should be of any value, because the time elapsing between the weighing of the person until death from cancer must average from twenty to thirty years, and in many cases be well over fifty years.

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* From the Huntington Memorial Hospital and Department of Surgery, Harvard Medical School. This work was supported by an anonymous gift to the Harvard Medical School.

1. Jackson, Henry, Jr.: Recent Advances in the Biology of Cancer, New England J. Med. **201**:294, 1929.

2. Hoffman, F. L.: The Mortality of Cancer Throughout the World, Newark, N. J., Prudential Press, 1915.

3. Jackson, D. L.: Carbohydrate Metabolism in Cancer, Texas State J. Med. **24**:622, 1929.

Another aspect of this general problem, and one that it is possible to study from data in the hospital records, is to determine whether there is a relationship between the patient's weight and the prognosis of the disease. This, of course, is quite different from trying to obtain a cure by diet or to show that there is a relation between weight and incidence.

METHOD OF STUDY

It seemed well to start the study with single clinical and pathologic entities in which a single form of treatment was used. At the Huntington Hospital, cases of carcinoma of the cervix and of the uterus are most suitable for study with respect to many factors. Dr. George A. Leland has had patients with these conditions under his care since 1920, and those with carcinoma of the cervix have been treated with radium rather than by surgical measures or mixed treatment. He has just completed a thorough survey of these cases from the opening of the hospital through the year 1924, and the percentage in which there was a complete five year follow-up is high. Beginning in 1920, a record of the patient's weight may be found in most instances. Therefore, this study was limited to the five year period from 1920 to 1924. Unfortunately, no records were made of the patient's height, so that conclusions as to the degree of obesity of a given patient cannot be drawn too accurately.

In preparing the tables, the following standards of suitability were followed:

1. Presence of clinical carcinoma of the cervix or of the uterus, proved by pathologic examination, at the time of the first treatment with radium.
2. Record of the patient's weight on first entry.
3. Known survival for five years or death from clinical cancer in less than five years.
4. Treatment with radium.[4]
5. No subsequent radical operation.

In order to adhere strictly to these standards, it was necessary to eliminate about half of the cases with a diagnosis of carcinoma of the cervix or of the uterus, according to the files in the hospital. These eliminations were made largely for three reasons, in the following order of frequency: (1) no record of weight; (2) no pathologic report or (3) patient lost from observation. (There were only twenty-four cases lost from observation that were otherwise suitable.) The cases without record of weight or those that were not followed could not be used.

The satisfactory cases were separated into two groups and each studied separately. These groups consist of two distinct pathologic enti-

4. The methods of treatment used may be found in G. A. Leland's "Radium Treatment in Cancer of the Female Pelvis" (Physical Therap. **48**:281 [June] 1930).

ties: (1) epidermoid carcinoma of the cervix (475 cases) and (2) adenocarcinoma of the cervix and of the uterus (79 cases). Cases of adeno-acanthoma, carcinoma simplex or colloid carcinoma were not included in any group. There were only 15 of these, and in all the outcome was fatal.

Patients surviving five years were arbitrarily considered as cured. These patients were again classified as to the results after the five year period (column *L* on the charts) as follows: (1) those who died from carcinoma or were alive but diseased and (2) those who were living and not diseased or who had died from other causes.

EPIDERMOID CANCER OF CERVIX

Relation of Weight to Survival.—From the total of over 900 cases of epidermoid carcinoma of the cervix, 475 were suitable for study.

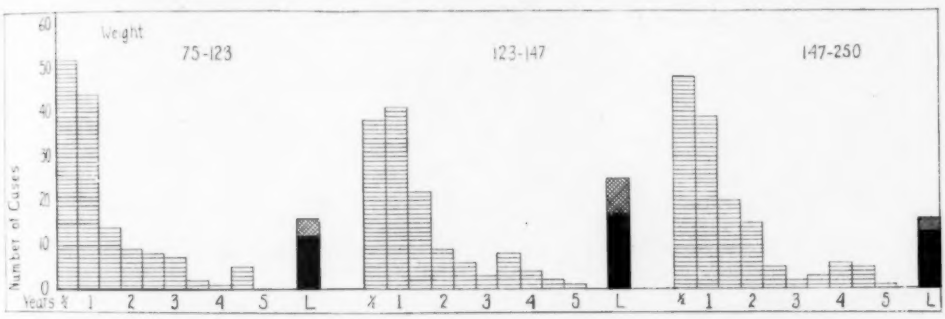

Chart 1.—Length of survival in six month periods of 475 persons with epidermoid carcinoma of the cervix. The patients are divided into three equal groups according to weight. In this and the following charts, the area composed of straight lines indicates those patients who died in less than five years; the crossed area, those who died from the disease or were alive and diseased after the five year period; the black area, those who were alive and well or who died from causes other than the disease after the five year period.

These were tabulated and divided in various ways according to weight. Whatever the method of division, the same general tendency will be seen.

Chart 1 shows the length of survival in six month periods in the cases divided into three equal groups according to weight.

Chart 2 is similar, showing the cases divided into five equal groups. It will be seen at once that the proportion of cases in which the patients survived five years is much higher among those of normal weight than among those whose weight was above or below normal.

Results shown in the foregoing manner would be difficult to compare with results from other clinics or from other groups of cases in our clinic because the points of division of other cases into equal fifths or

equal thirds might not correspond to these. The same cases are therefore presented in another manner in table 1. Here the cases are divided according to weight; 20 pound (9 Kg.) intervals are used in the groups from 75 to 174 pounds (34 to 78.9 Kg.), while weights above 174 pounds are included in a single group. In this table the time of survival of patients who did not live for five years is omitted, and only the total number of cases, the number of survivals and percentage of survivals are presented. Again, however, the survivals are subdivided into two

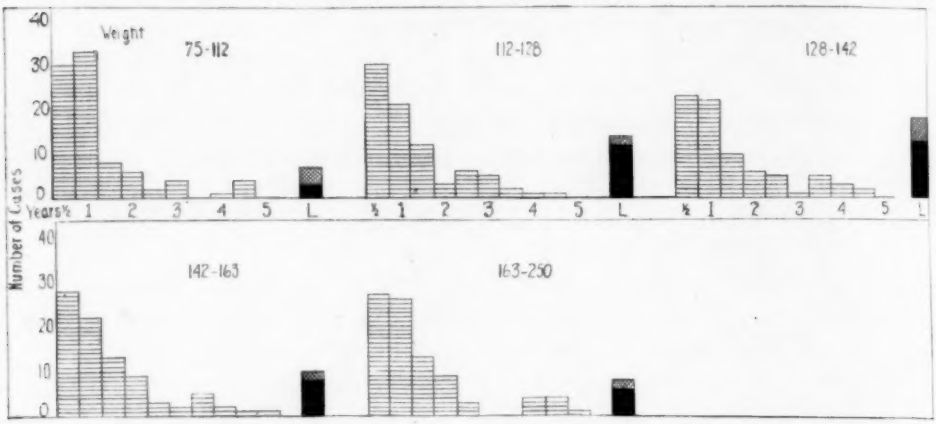

Chart 2.—Same as chart 1, except that the cases are divided into five equal groups.

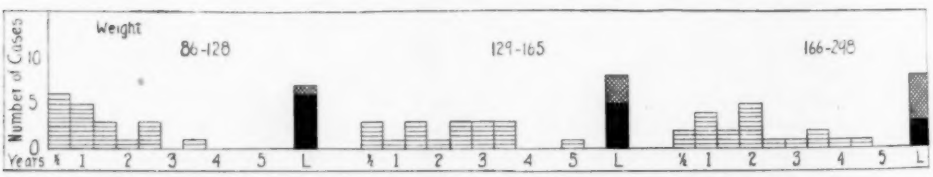

Chart 3.—Length of survival in seventy-nine persons with adenocarcinoma of the cervix.

classes as previously. The apparent discrepancies between this table and the preceding charts will be discussed later.

ADENOCARCINOMA OF THE CERVIX AND FUNDUS

For the purpose of this study the cases of adenocarcinoma of the cervix and of the fundus were grouped together. This does not mean necessarily that a single pathologic entity is being dealt with, but it was done purely for practical reasons. The first reason is that either group alone is too small to be at all significant statistically; the second, that it is not always possible to tell from the record whether the disease orig-

inated in one or the other part of the uterus. There were 39 cases of adenocarcinoma of the cervix and 40 cases of adenocarcinoma of the fundus. Chart 3 shows the 79 cases divided into thirds and presented in the same form in which chart 1 presents the cases of epidermoid carcinoma. It will be seen at once that the median weight in these cases

TABLE 1.—*Data on Four Hundred and Seventy-Five Cases of Epidermoid Carcinoma of the Cervix* *

Weight, Pounds	Number of Cases	5 Year Survival With Disease		5 Year Survival Without Disease		Total 5 Year Survival	
		Cases	Per Cent	Cases	Per Cent	Cases	Per Cent
75-94	21	1	5.0	..	....	1	**5.0**
95-114	90	3	3.3	6	6.6	9	**10.0**
115-134	127	2	1.5	14	11.0	16	**12.5**
135-154	113	6	5.0	11	10.0	17	**15.0**
155-174	59	3	5.0	7	12.0	10	**17.0**
175+	65	..	...	4	6.0	4	**6.0**

* These cases are divided into groups with weight divisions of 20 pounds each.

TABLE 2.—*Data on Seventy-Nine Cases of Adenocarcinoma of the Cervix and of the Uterus* *

Weight, Pounds	Number of Cases	5 Year Survival With Disease		5 Year Survival Without Disease		Total 5 Year Survival	
		Cases	Per Cent	Cases	Per Cent	Cases	Per Cent
75–123	21	..	....	4	19.0	4	**19**
123–147	21	2	9.0	5	24.0	7	**33**
147+	37	7	18.5	5	13.5	12	**32**

* These cases are divided into three groups corresponding to the weights at which the epidermoid groups were divided into equal thirds in chart 1.

TABLE 3.—*Data on Seventy-Nine Cases of Adenocarcinoma of the Cervix and Uterus* *

Weight, Pounds	Number of Cases	5 Year Survival With Disease		5 Year Survival Without Disease		Total 5 Year Survival	
		Cases	Per Cent	Cases	Per Cent	Cases	Per Cent
75-94	1	..	..	1	100	1	**100**
95-114	13	..	..	2	15	2	**15**
115-134	19	1	5	3	16	4	**21**
135-154	14	2	14	3	21	5	**35**
155-174	9	1	11	2	22	3	**33**
175+	23	5	21	3	13	8	**35**

* These cases are divided by weight into groups at 20 pound intervals.

is much higher than in the group of epidermoid carcinoma, and that, therefore, the dividing line between the upper and middle groups is 18 pounds (8.2 Kg.) higher. Therefore, table 2 has been made up to show a division in these cases at the same weights at which the cases of epidermoid carcinoma were divided.

Table 3 represents another tabulation of these cases and is made on exactly the same basis as table 1. The cases are listed in 20 pound divisions.

COMMENT

Statistics.—In work of this type it is fundamental to know whether there is any possibility that the statistics presented actually show what they appear to demonstrate. Fallacious findings may be due to accidental or systematic sources of error. By the former are meant especially errors due purely to chance. This type of error is gradually reduced as the series increases in size. Dr. E. B. Wilson informed me that a series of cases in which 57 are differentiated from a group of 475 and then systematically divided into from three to five groups is large enough to make the possibility of error due to chance very small. On the other hand, the second group presented, the adenocarcinomas, is so small that chance can easily account for the apparent differences. Every effort was made to eliminate systematic errors by rigidity in the selection of cases, so that selection was purely automatic on the basis of the records. None of the cases have ever been seen by me. As a partial check on the work, a group of cases was reviewed that was eliminated from the preceding tables for causes other than that the patient was lost from observation, there was no record of weight or that treatment had not been given. These cases included those in which the patients showed clinical cancer, but no pathologic report had been made, cases in which the patients had been operated on elsewhere and positive pathologic reports made, after which they were sent to this hospital for prophylactic radiation, and cases in which the patients had had radical operations performed elsewhere after receiving treatment with radium in this hospital. There were 78 of these cases, tabulation of which showed exactly the same tendency toward the grouping of cures in the patients of middle weight as in the proved cases of epidermoid carcinoma. This checks against the possibility that these eliminations changed the results in any way.

Epidermoid Carcinoma of the Cervix.—Whether these cases are presented in the form of a division into equal thirds, equal fifths or unequal division according to 20 pound intervals, it is definitely shown that the greatest percentage of cures occurred in the patients who were of middle weight.

An apparent discrepancy between the results in chart 2 and in table 1 lies in the fact that the greatest percentage of cures occurs at different points, higher in table 1 than in chart 2. Which form of presentation gives the truest picture cannot be stated.

Whatever form is used for tabulation, there is no doubt that the prognosis is more favorable for the more normal patients than for the fat or the thin ones. The obvious explanation for the unfavorable prognosis in the cases in which the patients are very thin is the increased proportion of patients who have lost weight and are in the terminal stages of the disease. This I believe to be true and shall not discuss further.

The reasons for the poor results in fat patients are, however, not so obvious. One factor probably is the purely mechanical difficulty of applying radium accurately. In making any explanations beyond this, one has to enter the realms of pure speculation. Does the fat person have less resistance, or does the malignant condition find a more fertile field for growth?

Adenocarcinoma of the Uterus and of the Cervix.—All three tabulations of these cases show approximately the same results, and results that are different from those shown for the cases of epidermoid carcinoma. If the patients who survived for five years are considered as a whole, there is no difference in the groups of different weights. It is striking, however, that in the group of patients who were overweight so many who were considered cured were alive and diseased or were dead from the disease after the five year period that the patients actually alive and well for more than five years were hardly 50 per cent of the corresponding number in the patients who were of middle weight.

The series of patients with adenocarcinoma is so small that these findings may be entirely fallacious for this reason alone. Another reason why these cases cannot be compared with those of epidermoid carcinoma of the cervix has to do with the indications for treatment. In the latter group, radium was used in all cases no matter how favorable the condition was for operation. On the other hand, half of the cases of adenocarcinoma were of the fundus. If the disease was not too extensive and the patients were otherwise in good condition, they were operated on and are therefore ineligible for this series. Obesity was, however, the most common contraindication to operation in the less advanced cases of adenocarcinoma of the fundus. This basis of selection has led to two probable results: 1. The patients with adenocarcinoma are heavier as a group than those with epidermoid carcinoma. (Of course, adenocarcinoma may be a disease that occurs more frequently in heavy people, but this cannot be determined from any data available.) 2. In the majority of cases the adenocarcinomas were less advanced in obese persons than in thin persons or in those of moderate weight.

SUMMARY AND CONCLUSIONS

1. Study of 475 proved cases of epidermoid carcinoma of the cervix in which treatment with radium was used shows that the percentage of cures of patients with approximately normal weight was twice that of patients who were much over or much under normal weight.

2. A group of seventy-nine patients with adenocarcinoma of the cervix or fundus of the uterus was similarly studied, and a definite but less marked relationship of weight to prognosis was found.

EXPERIMENTAL ESOPHAGEAL OBSTRUCTION*

CARL A. DRAGSTEDT, M.D.
AND
RALPH B. MULLENIX, M.S.
CHICAGO

As has been suggested by many workers, in cases of high intestinal obstruction there are two fairly well defined groups of factors that probably operate in the pathogenesis of the untoward symptoms that develop. Dehydration, with loss of chlorides and other constituents of gastro-intestinal and pancreatic secretions either by vomiting or by failure of reabsorption, on the one hand, and toxemia from the absorption of toxic substances from the intestinal lumen, on the other hand, may operate more or less independently at times, but probably more often concurrently and with a reciprocal influence in the usual case.

In an analysis of some of the factors concerned in intestinal obstruction, Haden and Orr,[1] in 1923, produced various types of experimental obstruction of the cardia and esophagus and reported that severe toxemia with rapid death resulted. They ascribed the symptoms and death to the same causes that were thought by them to be operative in obstruction of the gastro-intestinal tract at lower levels. Both the observations and the conclusion seemed to us to be difficult to explain. In the first place, it is well known that many patients survive obstructions of the esophagus either from stricture or from new growth without the development of fulminating symptoms related to the obstruction per se. In the second place, it is difficult to appreciate adequate mechanisms for either the loss of important gastro-intestinal secretions or the development of toxemia. Vomiting cannot occur, and it seems improbable that sufficient disturbance of the gastro-intestinal tract would develop to prevent reabsorption of the juices secreted. Similarly, the factors apparently necessary for the formation and absorption of toxic substances, such as distention, strangulation or impairment of the blood supply, are not established.

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* From the Department of Physiology and Pharmacology, Northwestern University Medical School.

1. Haden, R. L., and Orr, T. G.: Chemical Changes in the Blood of the Dog After Obstruction of the Esophagus and Cardiac End of the Stomach, J. Exper. Med. **38**:477, 1923.

In 1930, Andrus and Donnelly [2] corroborated the observations of Haden and Orr that toxemia and rapid death result from obstruction of the esophagus. They offered the suggestion that the loss of saliva might play a rôle in the lethal outcome, apparently disregarding the fact that many laboratory workers have succeeded in keeping dogs alive with an esophageal fistula in the repetition of sham feeding experiments such as Pavlov originated. In 1928, Haden and Orr [3] reported further experiments on obstruction of the cardia, in which they again emphasized the marked toxemia that develops. This belief was apparently concurred in by Wangensteen and Chunn,[4] although they commented on the facts that vomiting could not occur and that the factor of absorption was reduced to a minimum.

Because of the seeming conflict between these reports and the clinical experience with esophageal obstruction, and because they seemed to nullify many of the arguments with respect to the interpretation of the pathogenesis of symptoms in acute intestinal obstruction, we were stimulated to perform a number of experiments on esophageal obstruction which we are reporting at this time. All the experiments were performed on dogs. The surgical procedures were carried out under aseptic precautions and under general anesthesia with either ether or pentobarbital sodium.

EXPERIMENTAL WORK

1. *Simple Obstruction by Means of a Ligature.*[5]—In ten dogs the esophagus was obstructed in the neck by means of a stout ligature. Six dogs died in twenty, thirty-three, thirty-six, forty, sixty and seventy-four hours, showing at autopsy well marked pneumonia. Four dogs died in sixty-four, sixty-five, seventy-four and eighty-four hours, showing at autopsy no pneumonia, but marked cervical cellulitis and mediastinitis, apparently due to cutting through of the ligature with consequent leakage. Blood chlorides were followed in all the dogs. In the group with pneumonia there was an average fall of 83 mg. per hundred cubic centimeters of blood. In the remaining four animals, there was an average fall of 32 mg.

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2. Andrus, W. D., and Donnelly, J. L.: Effects of Certain Operations on the Esophagus of the Dog: Including Esophageal Obstruction and Complete Esophageal Fistula, Arch. Surg. **20**:1 (Jan.) 1930.

3. Haden, R. L., and Orr, T. G.: The Effect of Sodium Chloride on the Chemical Changes in the Blood of the Dog After Obstruction of the Cardiac End of the Stomach, J. Exper. Med. **48**:627, 1928.

4. Wangensteen, O. H., and Chunn, S. S.: Studies in Intestinal Obstruction, Arch. Surg. **16**:1242 (June) 1928.

5. The experiments in this group were done by one of us (Dr. Dragstedt) and Dr. John F. Huffman during 1927 and 1928.

In five dogs the esophagus was obstructed in the chest about 4 cm. above the diaphragm by means of a stout ligature. Three dogs died in twenty-seven, thirty-six and thirty-nine hours, showing at autopsy well marked pneumonia. Two dogs died in forty-eight and seventy-two hours, showing mediastinitis apparently due to cutting through of the ligature with consequent leakage. In the group with pneumonia the average fall in blood chlorides was 50 mg. per hundred cubic centimeters of blood. In the other two animals there was no fall in blood chlorides. It is thus apparent that the fall in blood chlorides is not comparable to that seen in acute intestinal obstruction and is no greater, in our opinion, than can be accounted for by the pneumonia.[6]

2. *Closed Loop Obstruction of the Esophagus.*—Closed loops of the esophagus were made in six dogs by ligating the esophagus just above the diaphragm in the chest, sectioning the esophagus in the neck, inverting the lower end and bringing out the upper end through the incision as a fistula. The animals died in thirty, forty-eight, sixty, sixty, seventy-four and seventy-six hours. At autopsy, two showed cellulitis of the neck but no leakage from the loop. The remaining four all showed mediastinitis subsequent to distention of the loop and leakage from one or both ends. It was noted, however, that none of the dogs had pneumonia. The significance of this observation will be referred to later.

3. *Simple Obstruction of the Exteriorized Esophagus.*—As 60 per cent of the animals in group 1 showed pneumonia at autopsy and none of the animals in group 2 developed pneumonia, it seemed possible that the pneumonia in group 1 was due to aspiration, which was prevented from occurring in group 2 by having the distal end of the oral segment of the esophagus drained to the outside. As these operations were done in one stage under general anesthesia, it was desired to exclude the factor of anesthesia in the development of the pneumonia.

In ten dogs the esophagus was exteriorized in the neck by freeing it for a considerable distance and suturing the muscles and skin of the neck beneath it. The technic of the operation we have described elsewhere.[7] Ten days later the exteriorized esophagus was simply obstructed by means of a ligature or a rubber-jawed clamp. In the five dogs with the ligature obstruction, immediate swallowing efforts with profuse salivation occurred, and the animals died in from eighteen to thirty hours, showing in every instance at autopsy marked con-

6. Snapper, J.: Ueber den Zusammenhang zwischen Funktion der Nieren und Chlorretention bei fieberhaften Krankheiten, Deutsches Arch. f. klin. Med. **111**: 430, 1913.

7. Dragstedt, C. A., and Mullenix, R. B.: Experimental Esophageal Fistula, J. Lab. & Clin. Med. **16**:591, 1931.

solidation of the lungs. In five dogs the obstruction was made by gradually tightening a metal clamp with rubber-covered jaws around the esophagus until it was occluded. In one of these dogs swallowing efforts were promptly made, and the animal died in twenty-eight hours, showing pneumonia at autopsy. The remaining four animals survived indefinitely, the clamp cutting through and establishing a fistula in three, four, four and six days, respectively, after which they were fed through the esophageal fistula. Until the establishment of the fistula, 500 cc. of 5 per cent dextrose was administered daily.

4. *Esophageal Obstruction After Preliminary Esophageal Fistula.*—From group 3 it was apparent that when the operative hazard incurred in obstructing the esophagus was eliminated by obstructing it some time after it had been exteriorized, there was still the hazard of 50 per cent or more animals developing an aspiration pneumonia, but in those animals that escaped the pneumonia there was no toxemia incident to the obstruction and they could survive until the obstruction relieved itself by the cutting through of the clamp. As we desired to produce a complete permanent obstruction, and as it seemed from the experiments in groups 2 and 3 that aspiration pneumonia would be precluded by preliminary esophageal fistula, we operated to obstruct the esophagus in a number of animals after first preparing a fistula of the esophagus in the neck. The fistula was prepared by the two-stage procedure described elsewhere,[7] and consisted, in brief, of exteriorizing a portion of the esophagus in the neck and some time later incising the anterior wall of the exteriorized portion.

In six animals the obstruction was made intra-abdominally by sectioning the cardia of the stomach and inverting both ends, from five to ten days after the preparation of the fistula in the neck. A gold-plated cannula was inserted into the stomach at this operation for purposes of feeding. Five of the animals died in from two to seven days. In four of these, definite cause for death was found post mortem, hemorrhage and peritonitis giving evidence of technical failure of the operation. In one no evident cause for death was found post mortem, but we are not inclined to attribute the death to toxemia from the obstruction as it is well known that the trauma and shock associated with sectioning both vagi below the diaphragm and the necessary stretching of the cardia involved in the operation are of serious significance. One animal, in which the operative procedures were carried out more skilfully, survived and did well until the eighth day, when it contracted distemper, and died on the eleventh day.

As we thought that possibly the operative hazard might be lessened by obstructing the esophagus intrathoracically above the diaphragm, this was done in ten dogs. Pentobarbital sodium was used as the

anesthetic in these animals, and artificial respiration was maintained by positive pressure insufflation of air through a catheter in the trachea. The incision was made through the eighth interspace on the left side. In eight animals the esophagus was carefully freed in its lower portion, the branches of the vagi being separated as far as possible. The esophagus was then sectioned with a knife about 3 cm. above the diaphragm and both ends were inverted and sutured with silk. The dogs died in from three to seven days, showing at postmortem examination varying degrees of empyema and mediastinitis. As we were convinced that we could not prevent a certain amount of soiling with subsequent bacterial infection by using the knife to section the esophagus, in the remaining two dogs the esophagus was sectioned with the cautery. The first one of these survived and remained well until the tenth day, when a subcutaneous infection developed, apparently induced by a subcutaneous administration of 5 per cent dextrose, and the dog died on the thirteenth day. At autopsy there was no empyema, pneumonia or mediastinitis, and the sutured ends of the esophagus were firmly closed. The second dog survived, and, in spite of a mild distemper and subcutaneous infection, it has made a good recovery and is now alive and doing well one month after the operation. Beginning on the second postoperative day, water was administered through the gastric cannula; on the fourth day milk was added, and since the seventh day a ration of milk, bread, eggs and ground steak has been fed.

COMMENT

It was apparent early in the work that there is a considerable surgical hazard in operative procedures on the esophagus. In such a relatively simple procedure as making a fistula in the neck, there is a mortality due to infection of over 50 per cent if the operation is done at one stage. That clinical surgeons have appreciated this is borne out by numerous papers and is well expressed in the review of esophageal surgery by Saint.[8] It was necessary, therefore, to separate and distinguish this hazard from the physiologic consequences of the obstruction. This can readily be done by first exteriorizing the esophagus in the neck, after which it can be obstructed without any surgical hazard whatever. When the obstruction was made by tying a stout ligature around the exteriorized portion, violent swallowing efforts immediately began, attended by profuse salivation, and the animals died within thirty-six hours, showing at postmortem examination an aspiration pneumonia with the lungs appearing the same as in a drowned person. When the obstruction was made with a rubber-jawed clamp, such swallowing efforts did not occur in four of the five instances, and the animals survived

8. Saint, J. H.: Surgery of the Esophagus, Arch. Surg. **19**:53 (July) 1929.

indefinitely and without any special treatment, the clamp cutting through by the third to the sixth day, thus establishing a fistula. With the latter procedure there was apparently not the same sensory stimulation provoking swallowing efforts as there was when the ligature was used.

These experiments demonstrated that in addition to the surgical hazard of operative procedures on the esophagus there was also a considerable likelihood of aspiration pneumonia. They also demonstrated that an obstruction could be produced and exist for from three to six days without producing any toxemia in the animals that escaped the pneumonia. This is also borne out by the experiments of Allen, published in 1925,[9] who successfully temporarily obstructed the esophagus in both the neck and the chest by ligating beneath a circular patch of fascia lata so that, as the ligature cut through the esophagus, leakage was prevented by the fascial transplant. He did not mention whether or not any of his animals developed pneumonia.

The experiments in group 4 again emphasized the grave risk from infection experienced in operations in which the lumen of the esophagus is opened. The last two experiments, we believe, however, prove that a complete permanent obstruction can be produced if the surgical technic is satisfactory, without the development of toxemia or loss of gastrointestinal secretions, and that there is, therefore, no essential discrepancy between the physiologic mechanisms of the dog and of man.

CONCLUSIONS

There is no toxemia specifically related to esophageal obstruction in dogs, and dogs can survive indefinitely a permanent complete obstruction without special therapeutic measures.

Acute esophageal obstruction in dogs leads to the development of an aspiration pneumonia in a high percentage of cases. The operative procedures employed in producing acute esophageal obstruction are attended with an extreme surgical hazard from infection. These facts are to be appreciated and distinguished from the physiologic consequences of the obstruction itself.

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9. Allen, D. S.: Further Experimental Reconstruction of the Esophagus with Autogenous Fascia Lata Transplants, Arch. Surg. **10**:374 (Jan.) 1925.

FORTY-SIXTH REPORT OF PROGRESS IN ORTHOPEDIC SURGERY*

PHILIP D. WILSON, M.D.

LLOYD T. BROWN, M.D.

M. N. SMITH-PETERSEN, M.D.

JOHN G. KUHNS, M.D.
AND
EDWIN F. CAVE, M.D.
BOSTON

RALPH K. GHORMLEY, M.D.
ROCHESTER, MINN.

MURRAY S. DANFORTH, M.D.
PROVIDENCE, R. I.

GEORGE PERKINS
LONDON, ENGLAND

ARTHUR VAN DESSEL, M.D.
LOUVAIN, BELGIUM
AND
C. HERMANN BUCHOLZ, M.D.
HALLE, GERMANY

CONGENITAL DEFORMITIES

Cervical Rib.—Telford and Stopford [1] recorded three cases of cervical rib in which the prominent symptoms were due to thrombotic obliteration of the arteries of the arm. These cases resembled one another in their gradual onset, the initial pallor, coldness and numbness of the limb, the occurrence of gangrene and the cessation of all pulse at a point about the junction of the axillary and brachial arteries, and finally in their complete recovery following the removal of a cervical rib. In no case was the subclavian artery found at operation to be constricted. Telford and Stopford believed the thrombosis to be due to irritation of the sympathetic nerve fibers supplying the peripheral arteries. They demonstrated by histologic sections that these sympathetic nerve fibers lie in the lowest trunk of the brachial plexus, and are therefore liable to compression by a cervical rib. They assumed that the compression irritated the sympathetic fibers and the irritation

* This Report of Progress is based on a review of 200 articles selected from 399 titles dealing with orthopedic surgery appearing in the medical literature between April 11, 1931, and June 27, 1931, supplemented by a few selected articles of older date. Only those papers that seem to represent progress have been selected for review.

1. Telford, E. D., and Stopford, J. S. B.: Brit. J. Surg. **1**:557, 1931.

caused spasm of the walls of the arteries; this spasm embarrassed the vasovasorum, which led to deterioration in the arterial walls and consequent thrombosis. In support of this hypothesis they produced experimentally a similar thrombosis by injecting ergot into cockerels.

Congenital Dislocation of the Hip.—Lowman [2] reported a procedure which he called "the double leaf shelf operation." A new acetabulum was made as low on the ilium as possible. Two rectangular bone flaps were then cut and turned down, one forward and downward, the other backward and downward. The adjacent corners of these flaps were sutured together with chromic catgut, and bone chips, obtained in large part from the removal of the upper margin of the original acetabulum, were packed back of the flaps. A check suture tied to the greater trochanter held the flaps in place.

[ED. NOTE.—The "shelf operation," in this country at least, is well established. The choice of operation is not so important as the careful performance of whatever operation is chosen. Lowman's operation offers another choice. We question the advisability of suturing the shelf to the trochanter.]

A Simple Modification of Putti's Splint for the Early Treatment for Congenital Dislocation of the Hips.—Coonse [3] combined traction with abduction in using Putti's splint for congenital dislocation of the hip in children 1 to 4 or 5 years of age. Skin traction was used, and the traction ropes were carried over large wooden pulleys at the ends of the splint and fastened to a spring balance scale. Usually 5 or 6 pounds of traction were sufficient to accomplish reduction.

BACK PAIN AND SCIATIC PAIN

Studies of the Nucleus Pulposa.—Geist,[4] basing his paper on the work of Schmorl, noted the frequent affection of the intervertebral disk and nucleus pulposa by disease and injury. The intervertebral disk comprised about one fourth of the movable parts of the spine, 40 per cent in the cervical region, 33.3 per cent in the lumbar and 20 per cent in the dorsal regions. Pathologic conditions of the spine in which the disk played a prominent part were: (1) prolapse of the disk, (2) adolescent kyphosis, (3) poor posture, (4) osteoporosis, (5) senile changes and (6) chronic spondylitis, which was always preceded by changes in the disk usually of the type of cartilage prolapse. In the following conditions the pathologic process of the disk was of secondary nature: 1. In fractures of the vertebrae the disk was often injured. 2. In injury without fracture there was frequently prolapse of the disk. 3. In sco-

2. Lowman, C. L.: J. Bone & Joint Surg. **13**:511, 1931.
3. Coonse, G. K.: J. Bone & Joint Surg. **13**:602, 1931.
4. Geist, E. S.: Intervertebral Disk, J. A. M. A. **96**:1676 (May 16) 1931.

liosis the rôle of the disk had not yet been determined, but it probably played a part. 4. In osteomalacia there were softening of the bone and consequent ballooning of the disk. 5. In tuberculosis of the spine the disk frequently disappeared early in the disease. 6. In osteomyelitis the disk was rapidly destroyed. 7. Syphilis of the spine was characterized by bony density and cartilage prolapse. 8. Charcot spine was characterized by marked hyperostoses and disappearance of the disk. 9. Carcinoma, or other destructive bone lesions, often showed cartilage prolapse into the substance of the bone.

Sashin [5] discussed the significance of intervertebral disk extension into the adjacent vertebral bodies. He had studied the condition, roentgenologically and at autopsy, and felt that in the great majority of cases trauma, either acute or repeated over a long period of time as in the case of men employed at hard labor, was the etiologic factor. In some instances the lesion might be caused by degenerative changes of the cartilaginous plate of the vertebral body by the expansile disk, as seen in osteomalacia, tumor metastases and osteoporosis, when the resistance offered by the vertebra was lowered. The author gave full credit for the knowledge of intervertebral disk disease to Schmorl, who first described it in 1926.

Fibrocartilaginous Nodules of Intervertebral Disk Causing Compression of the Spinal Cord.—Alajouanine and Petit-Dutaillis [6] made a study of the anatomy and pathogenesis of the new variety of radiculomedullary compression caused by fibrocartilaginous nodules of the posterior face of the intervertebral disk. They found a report of twenty-one cases in the literature of which in ten cases the location of the tumor was in the lumbar region, in three cases in the dorsal region and in seven cases in the cervical region. They reported two cases of their own, both with unilateral lumbosciatic pain of from three to four years' duration before motor disturbances appeared finally leading to the diagnosis. In both cases the tumor was situated in the lower lumbar region. They emphasized the fact that the nodules were almost invariably in a lateral rather than a median location.

Discussing the significance of these tumors, the authors stated that in view of the studies of Schmorl and Androe it seemed probable that the starting point of the lesion was a herniation of the nucleus pulposus through a fissure in the posterior part of the disk. To account for the herniation of the disk there were the traumatic theory and the theory of previous alterations of the nucleus and of the disk itself.

[ED. NOTE.—The editors agree that the intervertebral disk is coming into its own as a recognized factor in pathologic conditions of the spine.

5. Sashin, D.: Intervertebral Disk Extensions Into Vertebral Bodies and Spinal Canal, Arch. Surg. **22**:527 (April) 1931.

6. Alajouanine, T., and Petit-Dutaillis, D.: Presse méd. **38**:1657, 1930.

They wish to point out, however, that many variations of the disk probably occur that are without pathologic significance.]

A Groove for the Hypogastric Vessels.—Ferguson [7] pointed out that in the roentgenologic diagnosis of arthritis of the sacro-iliac joints error might arise when a groove in the ilium for the hypogastric vessels caused the inferior margin of the joint on the iliac side to appear sharply pointed, simulating the osteophytic outgrowth or lipping of arthritis. The prevalence of this groove was not generally recognized. A recent examination of the films of a hundred patients showed the groove present in some degree in eighty-six.

RICKETS AND CALCIUM METABOLISM

In a recent study of the replacement of the bone salts in healing scurvy, Salter and Aub [8] tried to determine whether or not the stores of lime salts in the bone were depleted at the same time that calcification of the growing portion was inhibited by lack of the antiscorbutic vitamin. At various stages of the disease in experimental animals the missing vitamin C was supplied in the form of orange juice and at the same time alizarin was injected so that the newly deposited bone salts would be stained red, and thus be easily detected. It was found that in healing scurvy the epiphyseal line was especially well marked, indicating a deposition of salts in the zone of rapid calcification. Furthermore, the cancellous tissue of the epiphysis and diaphysis was also stained, the pink trabeculae extending a short way into the medullary cavity. In addition, the shaft was colored by the dye distal to the epiphysis, although not as deeply red as were the trabeculae. On the other hand, the bones of animals receiving no source of vitamin C whatever remained unstained by the dye in spite of the fact that the experimental ration contained calcium and phosphorus.

In other words, scurvy was a derangement of calcification of the bones which affected the storage of bone salts in the trabecular tissue as well as the deposition in the epiphyseal region.

Fibrous Osteodystrophy in Experimental Hyperparathyroidism in Guinea-Pigs.—Jaffe and his co-workers [9] studied the effect of single and repeated doses of parathyroid extract in guinea-pigs. After a large single injection (20 units per hundred grams of weight) into young guinea-pigs, definite histologic changes were observed in six hours. Forty-

7. Ferguson, A. B.: J. Bone & Joint Surg. **13**:568, 1931.

8. Salter, W. T., and Aub, J. C.: Studies in Calcium and Phosphorus Metabolism: Deposition of Calcium in Bone in Healing Scorbutus, Arch. Path. **11**:380 (March) 1931.

9. Jaffe, H. L.; Bodansky, A., and Blair, J. E.: Fibrous Osteodystrophy (Osteitis Fibrosa) in Experimental Hyperparathyroidism of Guinea-Pigs, Arch. Path. **11**:207 (Feb.) 1931.

eight hours after injection the decalcification was so intense that the cortices of the ribs were fractured. Hemorrhage was observed in the marrow cavity; there were dilatation of the blood vessels and slight fibrosis of the marrow. There was cessation of all bone formation. The serum calcium tended to be higher from twenty-four to forty-eight hours after injection. The serum phosphorus remained within normal limits. When the guinea-pigs had fasted twenty-four or more hours before the injection there was a more definite rise in the serum calcium and also a rise in the phosphorus. In adult guinea-pigs there was no definite histologic change in the bones after single injections of parathyroid extract, although a rise in the serum calcium and phosphorus was observed. Repeated small doses of parathyroid extract in young pigs produced extensive fibrosis of the marrow and enlargement of the blood vessels, but no extensive formation of osteoid tissue. Larger doses had a more definite effect. Destructive lesions were seen in the bone marrow cavity with osteoid tissue formation in the healing process. Where the destructive lesion produced a sufficiently severe disturbance in the circulation, cyst formation resulted.

Ingestion of food was found to be an important factor in modifying the effect of the injection of parathyroid extract on the serum calcium and phosphorus. With small repeated doses, no definite rise in the calcium and phosphorus was observed. With large doses, the calcium was markedly elevated. The authors concluded that the changes produced in guinea-pigs were of the same order as those described for osteitis fibrosa without osteoid tissue in man.

NEOPLASMS

Metastatic Lesions of the Bones.—The analysis by Copeland [10] of 334 cases of metastatic lesions of the bone was one of the most comprehensive available. In the 1914 series of primary breast tumors there were 100 cases with bone metastasis. The bones most frequently involved were in order of frequency, the spine, pelvis, femur, skull, ribs and humerus. These lesions were most often multiple, being single in only one fourth of the cases. The most common type of lesion was an osteolytic lesion, while a sclerosing lesion was encountered only twice in the series. Roentgen therapy gave relief from the excrutiating pain, and in some instances definitely prolonged life. Of 1,040 cases of primary prostatic carcinoma, there were 134 with bony metastases. Only 50 per cent of the patients, however, were examined by the roentgen rays, and in only 25 per cent of this number were metastases found. The bones most frequently involved were the pelvis and vertebrae, and the metastatic lesions were all predominantly osteoplastic. Of 63 cases of hypernephroma, 22 showed bony metastases. The bones

10. Copeland, M. M.: Radiology **16**:198, 1931.

affected were in order of frequency, humerus, spine, femur, pelvis, ribs, bones of the feet, skull and sternum. It was to be noted that this was the highest incidence of bone involvement found in the entire series, there being bony metastases in 34.9 per cent of the cases, while in prostatic carcinoma it amounted to 12.8 per cent, in breast tumors to 5.2 per cent and in lung carcinoma to 16.6 per cent (four cases).

Neoplastic Lesions of the Upper End of the Humerus.—Major,[11] reviewing the neoplastic lesions of the upper end of the humerus, noted the following points:

1. The two most frequent osteolytic lesions did not require surgical intervention. They were the benign cyst and the malignant metastatic tumor the origin of which was elsewhere than the defect in the bone. The bone cyst was best left alone, or, if a pathologic fracture had occurred, treated by light roentgen radiation. The metastatic tumor was best treated in this late stage by heavy doses of deep roentgen radiation.

2. In all other cases in which the nature of the tumor was doubtful, the wise procedure seemed to be to put the arm at rest, administer roentgen therapy and obtain competent consultation on the roentgen films, rather than to do an immediate exploration for microscopic examination.

3. Resection in all malignant lesions of the upper end of the humerus offered as much for cure as did amputation and more for the patient, if cured.

Neoplastic Lesions of the Lower End of the Radius.—Oliver[12] studied fifty lesions of the lower end of the radius. Of the various neoplasms encountered in the radius, 90 per cent were in the lower end, and of these, thirty-five, or 70 per cent, were giant cell tumors. Other conditions were exostoses, 8 per cent; nonsuppurative osteomyelitis, 6 per cent, and bone cyst, 6 per cent. One case each, or 2 per cent, was noted of chondroma, chondrosarcoma, Ewing's tumor, osteolytic sarcoma and sclerosing sarcoma. All the patients with benign lesions had been cured, and of the patients with giant cell tumors 74 per cent had required only one treatment by curettement and thermal or chemical cauterization.

Tumors of the Os Calcis.—Of 1,740 tumors reviewed at the Johns Hopkins Surgical Pathological Department, Moore[13] found that 33, or 2 per cent, involved the os calcis. Exostosis was the most common type of new growth, occurring in 23 of the total number. All were treated by excision; cure was obtained in 20, and in 3 there was

11. Major, R. C.: Radiology **16**:224, 1931.
12. Oliver, R. L.: Radiology **16**:245, 1931.
13. Moore, J. R.: Radiology **16**:232, 1931.

recurrence. No malignant changes were observed in this series. Chondroma, giant cell tumor, chondrosarcoma and Ewing's tumor composed the remainder. No bone cysts, myeloma or metastatic tumors were found. Chondromas of the os calcis had to be treated in the same manner as chondromas of the long bones. They ought to be watched carefully with frequent roentgen examinations and the malignant possibilities borne in mind. If malignant changes became evident, they were to be treated as chondrosarcomas, and early amputation offered the only hope. The giant cell tumors, when recognized before the shell perforated, responded to curettage, and apparently had little tendency to recur. Excision or amputation might be necessary when the invasion of the soft part was extensive. Ewing's tumor offered little hope for life, judging by the two cases reported. However, in neither case was the diagnosis made before the end of twenty-four months. It is possible that with early diagnosis, amputation and roentgen therapy might have helped.

Nonsuppurative Osteomyelitis.—Cohn [14] studied 105 cases of nonsuppurative benign lesions of bone grouped under the diagnosis of Paget's disease, ossifying periostitis and nonsuppurating osteomyelitis. More than one bone was involved in 25 per cent of the cases; the remaining 75 per cent showed single lesions. The tibia and femur were each involved in about 20 per cent, the humerus in about 8 per cent, the skull in 5 per cent and the clavicle in 5 per cent. The remaining 35 per cent included lesions of the radius, ulna, fibula, metatarsal, metacarpal, carpal and tarsal bones, phalanges, ribs, vertebrae and jaw. In about 33 per cent the roentgen examination alone suggested that there was a question of sarcoma. There were four types of lesions: sclerosing, ossifying, destructive and osteoporotic, and four etiologic factors, viz., traumatic, syphilitic, posttyphoid, or postinfluenza and focal infections.

In respect to treatment, Cohn stated that "when the roentgen examination suggested sarcoma, and the tumor was radioresistant, and all foci of infection had been eliminated, exploratory operation was justified provided the tumor, if malignant, was operable."

Pathologic Lesions of the Bones of the Hands and Feet.—Kahn [15] studied 140 pathologic lesions of the small bones of the hands and feet. Of forty cases of exostoses, the os calcis was involved in 50 per cent. The youngest patient was 7 years of age and the oldest 65. The chondromas numbered forty-five, and were almost equally divided as to sex. The youngest patient was 6 years of age, and the oldest 80. In thirty cases the phalanges of the hands were involved; in seven the

14. Cohn, L. C.: Radiology **16**:187, 1931.

15. Kahn, M.: Radiology **16**:211, 1931.

metatarsals; in six the phalanges of the feet, and in two the tarsals. There were no cases of chondroma involving either the carpals or the metacarpals, an important diagnostic point in differentiating this lesion from benign giant cell tumor and bone cyst. This study emphasized the rarity of malignant disease in the small bones of the hands and the feet. If a lesion was single in character and central and osteolytic in type, a malignant condition could be practically ruled out. Periosteal, osteogenic sarcoma occurring as a single lesion in the small bones of the extremities was rare. It had been found in about 2 per cent of the cases, and was difficult to differentiate from the more frequent lesion of ossifying periostitis. The usual tumor encountered in the bones of the hands and feet contained cartilage, and was either an exostosis or a chondromyxoma, both of which could be safely treated as benign. Most of the remaining tumors belonged to the giant cell group, or to the closely related group of osteitis fibrosa or xanthoma, which also were typically benign. When metastatic nodules occurred in the small bones, their nature could be readily discerned by the presence of diffuse involvement of the remainder of the skeleton, which was always associated with their presence in the small bones.

[ED. NOTE.—The foregoing articles are a part of a group dedicated to Dr. J. C. Bloodgood by his pupils. They represent observations on material collected over many years, studied extensively, and followed up as accurately as possible. We therefore believe them to be of the utmost importance in leading toward a better knowledge of this most important subject, tumors of bone.]

Liposarcoma.—Stewart [16] reported what he believed to be three cases of primary liposarcoma of the bone. All occurred in young men. In each case there were multiple bony tumors loaded with fat cells, and there was no evidence of their having originated from endothelium. The author felt justified in reporting these tumors as liposarcomas in the hope of encouraging other pathologists to look for similar tumors.

Benign Giant Cell Tumor.—Dyke,[17] a pathologist, reported a case of benign giant cell tumor with a fatal termination after a clinical course of five years. The original lesion was in the patella; this was curetted. A local recurrence developed after three years, and the leg was amputated. Death took place one year later. At the autopsy metastases exhibiting the same structure as the primary tumor were demonstrated in the scalp, lung, kidneys and mediastinal and peritoneal lymph nodes. Photomicrographs of the primary and of the secondary tumors were given.

Orr had reported a second case. The leg was amputated in January, 1898, and death took place seven months later. Photomicrographs of

16. Stewart, F. W.: Am. J. Surg. **7**:87, 1931.
17. Dyke, S. C., and Orr, J. W.: J. Path. & Bact. **34**:259, 1931.

the primary tumor were unavailable, but those of the metastatic deposits in the lungs showed the typical picture of benign giant cell tumor.

POLIOMYELITIS

Experimental.—Harmon and his co-workers [18] described the preparalytic symptomatology in monkeys after intracerebral inoculation with the virus of poliomyelitis. In from five to eight days after inoculation the animal became passive or restless. This was followed by a period of excitement with cries and tremors in the muscles. With this there was a rise in temperature of from 1 to 3 degrees. Spinal fluid changes were found not earlier than forty-eight hours before paralysis. There was increased pressure, and globulin was found. There was very little change in the white blood count. The erythrocyte sedimentation rate was increased about twenty-four hours after the onset of the paralysis.

Missed Infections, Abortive and Relapsing Cases of Experimental Poliomyelitis.—Harmon and his co-workers [19] found that of 350 monkeys inoculated intracerebrally with the virus of poliomyelitis, 7 resisted infection and showed no symptoms, elevation of temperature or spinal fluid changes. Four of these animals later succumbed to a second inoculation. Other animals of the same incubation period, with preparalytic symptoms, rise in temperature, cerebrospinal fluid changes, but not paralysis, were classified as having abortive cases of experimental poliomyelitis. There were ten such cases. In nine reinoculation was done, and in five typical paralysis developed. The authors felt that such cases were better explained on the basis of a difference in susceptibility rather than as due to mild infection from an attenuated virus.

Dry Storage of Convalescents' Serum.—Lichtenstein [20] reported that at the Stockholm Epidemic Hospital, Sweden, the greater part of the store of serum for both prophylaxis and treatment had been kept in a dried form. The experience there showed that serum so stored retained its efficiency for a long time, two or three years at least.

TUBERCULOSIS

Tuberculous Spondylitis.—Doub and Badgley [21] recognized three types of tuberculous lesions of the spine, depending, they believed, on what particular part of the blood supply of the vertebrae was involved. The three types were, first, central involvement of the vertebral body;

18. Harmon, P. H.; Shaughnessy, H. J., and Gordon, F. B.: J. Prev. Med. **5**:115, 1931.

19. Harmon, P. H.; Shaughnessy, H. J., and Gordon, F. B.: J. Prev. Med. **5**:139, 1931.

20. Lichtenstein, A.: Dry Storage of Convalescents' Serum, J. A. M. A. **96**: 2102 (June 20) 1931.

21. Doub, H. P., and Badgley, C. E.: Am. J. Roentgenol. **25**:299, 1931.

second, primary involvement of the anterior portion of the vertebrae, and third, a type which they chose to call intervertebral articular tuberculosis.

They were particularly interested by this last type, and reported three such cases. They were difficult to diagnose clinically, because of the usual absence of physical findings, and the roentgenogram furnished the only evidence. An abscess might, however, be present before roentgen changes had occurred. There was usually definite narrowing of the intervertebral space, but the contour of the adjacent bodies might be preserved, and only later could erosion of the adjacent bodies be demonstrated.

Calcification of Tubercles by Means of Viosterol in Experimental Chronic Tuberculosis.—Spies [22] produced a chronic tuberculous infection in rabbits, and then gave large doses, from 2 to 5 cc., of viosterol every two to five days until the animals died. He observed a precipitation of calcium in the tubercles far greater in frequency and degree than in a series of control animals. The calcium deposit was almost entirely limited to the necrotic and caseous portion of the tubercle. The author suggested that smaller, nontoxic doses might be of value in the healing of tuberculous lesions.

Laminectomy in the Treatment for Pott's Paraplegia.—Basing their arguments on a personal experience with fifteen patients, Le Fort and Baudelot [23] defended the effectiveness of laminectomy in relieving paraplegia resulting from Pott's disease. In their fifteen patients there were no postoperative deaths, but three died from fifteen months to four years after the intervention. The results were described as excellent (normal gait) in two; good (walking possible) in two; moderate (improvement) in five; nil in two, and one case was too recent to classify. In four of the patients the paralysis was of the precocious type; in six of the delayed type. The operation had been performed in from four months to seven years after the onset. The authors emphasized particularly the frequency of bony compression of the spinal cord, this having been found in ten of eleven cases. In four of the cases no trace of external pachymeningitis had been found.

The authors had never found any evidence of compression from an abscess. Their operative results were also contrary to the traditional views; their four successful results had been observed in cases of delayed and progressive paralysis, usually considered incurable, while their failures had resulted entirely in the rapid and precocious type of paralysis.

The authors felt, however, that it was still very difficult to formulate the indications for operation. They thought that the high frequency

22. Spies, T. D.: Am. Rev. Tuberc. **28**:169, 1931.
23. Le Fort, R., and Baudelot: Scalpel, Liége, Jan. 10, 1931.

of bony compressions was an argument in favor of operation. They had decided to intervene in cases of sudden or rapidly developing paralysis if no improvement had been obtained at the end of one year, and in the delayed and progressive types at the end of six months. They concluded that the operation was relatively benign, and that its effectiveness had been demonstrated sufficiently to make it worthy of consideration more frequently than at present.

[ED. NOTE.—Le Fort and Baudelot's observations are of considerable interest, especially because they are in contradiction to the commonly held views. The high frequency of bony compression found naturally raises a question of the effectiveness of the preoperative treatment their patients had received, as we feel that bony compression can usually be prevented or relieved by measures directed toward obtaining hyperextension and thorough fixation of the spine. A finding in some of the editors' cases has been a tuberculoma, and this was not found in any of the cases reported by the foregoing authors. Their views will require confirmation before we can accept them completely.]

PYOGENIC INFECTIONS OF BONES

Maggot Treatment.—Baer [24] described the use of maggots, the larvae of the blow fly, in the treatment of eighty-nine patients with osteomyelitis. The maggots were grown under asceptic precautions, and were placed on the wound from twenty-four to forty-eight hours after operation had been performed. No dressing was required, but the wound was covered by a snug fitting wire screen cut to the size of the wound to prevent the maggots from crawling out. The maggots removed the dead tissue and much of the secretion from the wound. The reaction of the wounds became alkaline soon after the introduction of the maggots. The wounds were washed out every five days and new maggots introduced. Granulation tissue filled in rapidly, and the wounds usually were soon solidly healed. There had been a recurrence of the osteomyelitis in 5 per cent of the cases. Such recurrences generally consisted in the extrusion of small spicules of bone after which the wounds remained healed. In a small number of cases with open tuberculous abscesses, encouraging results were obtained by wide incision of the abscess followed by maggot treatment. The author felt that in addition to the scavenger action, the maggots exerted some biochemical effect on the wound which was inimical to bacterial growth and hastened tissue repair.

[ED. NOTE.—No form of treatment has created more widespread discussion in the last few years than Dr. Baer's maggot treatment for

24. Baer, W. S.: J. Bone & Joint Surg. **13**:438, 1931.

osteomyelitis. We hope that the untimely death of its author will not interfere with the continuation of experiments aiming to evaluate it properly. We are of the opinion, however, that technical difficulties will prevent any general adoption of it.]

An Evaluation of the Orr's Treatment for Osteomyelitis.—Kulowski [25] made a study of 130 cases of osteomyelitis, 16 case of suppurative arthritis and 9 of extensive soft tissue suppuration, with treatment by Orr's method, and drew highly eulogistic conclusions. He recommended the method for routine use, claiming that it was painless, that it reduced the period of hospitalization, that it insured good functional results, and that it brought about improvement in the general condition of the patient, minimized loss of limb, shortened the postoperative course and was suited to the rank and file of the profession. It was attended by an insignificant mortality rate.

[Ed. Note.—The article by Kulowski is thorough, and presents all of the arguments in favor of Orr's treatment. We believe that in the treatment for osteomyelitis thorough operation and adequate drainage are more important than the particular type of postoperative treatment employed.]

Epithelization of Bone Cavities and Calcification of Fibrous Marrow in Chronic Pyogenic Osteomyelitis.—Brunschwig [25a] reported a histopathologic study of three cases of chronic pyogenic osteomyelitis of over fifty years' duration, which showed:

1. Epithelization of the draining sinuses and partial to nearly complete epithelization of large cavities in the bone to which they led.

2. Calcification of fibrous marrow in cancellous bone bordering the cavities with infection and necrosis and partial or complete sequestration of these bony and calcified areas.

3. Calcification in fibrous marrow away from the epithelized cavities, surrounded and traversed by living bone.

As to treatment, two of the three patients died after amputation which had seemed advisable because of the inconvenience and pain caused by the lesion over a period of many years. In the third patient, in whom more conservative treatment was employed, the cavity being extensively effaced by removal of much of its walls and all sequestrums, foreign bodies and soft parts, no relief from pain or purulent discharge was obtained. The condition of the leg remained essentially the same as before operation. In fact, the cavity had become relined

25. Kulowski, J.: J. Bone & Joint Surg. **13**:538, 1931.

25a. Brunschwig, A.: Surg. Gynec. & Obst. **52**:759, 1931.

with epithelium at the end of two years, and there appeared to be no reduction in the amount of discharge.

[ED. NOTE.—The authors' studies confirm our opinion that extensive osteomyelitis of many years' duration does not lend itself to an operation for radical cure.]

(To be Concluded)